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DIAGNOSIS AND TREATMENT OF TUBERCULOSIS OF THE LARYNX AND CONTIGUOUS AREAS

GEORGE E. WILSON, M.D.

SARANAC LAKE, N. Y.

The title of this paper was chosen so as to allow the inclusion of a discussion of tuberculous lesions of the mouth, pharynx, middle ear, trachea and bronchi, as well as those of the larynx. The conclusions are based on impressions drawn from more than twenty years' experience in the treatment of tuberculosis—seventeen of those years in private practice and the other, early, years in institutions. The bulk of the work has been, of course, laryngeal, and the accompanying figures tabulate data on over 1,000 patients treated in private practice. Many more patients with this serious complication of pulmonary phthisis were seen in sanatoriums and hospitals, but data on them are not included in the tables. However, they have helped me to formulate my opinions and conclusions. Naturally, the number of lesions in the mouth and in the pharynx has been small in comparison. The gross number of lesions of the middle ear encountered through the years is large, although the percentage as related to the number of cases of pulmonary tuberculosis must be less than 5. Unfortunately I have not had time to go through the histories and select the cases in which there were such lesions and to compile statistics. Tuberculous tracheobronchitis has demanded increasing attention and study during the past four or five years, and I shall devote some part of this article to the bronchoscopic investigations of me and my associates and our experiences in this most interesting phase of the tuberculosis problem. Peculiarly, I have not encountered tuberculosis of the nasal passages, with 1 possible exception. The 1 case was that of a woman of 60 with an obscure nasal lesion involving the posterior part of an inferior turbinate, biopsy of which showed chronic inflammation with giant cells, but the pathologist was not certain of his diagnosis. Nasal tuberculosis has been reported by Vandervere [1] and

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1. Vandevere, W. E.: Some Experiences with Otolaryngological Conditions in the Tuberculous, Eye, Ear, Nose and Throat Monthly **16**:84-85 and 101 (April) 1937.

Terry.[2] Likewise, tuberculosis of the paranasal sinuses must be rare, and I have seen no such lesion in my own practice. Hersh [3] described a case of tuberculosis of the right maxillary sinus and gave a review of the literature, listing reports of such cases by Gleitsmann,[4] Coakley,[5] Kechwick [6] and others.

Tuberculosis of the larynx is a vanishing disease. The main factor in the decreased incidence is the extensively increasing use of collapse therapy, namely, pneumothorax (intrapleural and extrapleural), with pneumolysis when indicated, interruption of the phrenic nerve, thoracoplasty and, to a lesser extent, pneumoperitoneum, surgical implantation of paraffin packs and external application of thoracic shot bags. Laryngeal lesions seldom appear after such treatment is instituted, and if already present, usually improve as the pulmonary and the general condition show the beneficial effects, especially if sputum conversion is effected by the operative measures. Dworetzky [8] and also Cooper and Benson [9] have stressed these therapeutic methods in the treatment of laryngopulmonary tuberculosis. At the Trudeau Post-Graduate School of Tuberculosis it has been found increasingly difficult of recent years to corral a full quota of ambulant patients to demonstrate to our students all the different types of laryngeal lesions in the various stages of advancement. Of course there is no dearth of bedridden patients in whom the disease is far advanced. Early pulmonary diagnosis with immediate treatment is another cause of the prevention of a tuberculous deposit in the larynx. The intake of vitamin C—cod liver oil and tomato juice—has been advanced by McConkey [10] as a preventive factor.

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2. Terry, G. H. B.: Upper Respiratory Tuberculous Complications of Pulmonary Tuberculosis, South. M. J. **27**:626-630 (July) 1934.

3. Hersh, J. H.: Tuberculosis of the Maxillary Sinus, Arch. Otolaryng. **28**: 987-993 (Dec.) 1938.

4. Gleitsmann, J. W.: Tuberculosis of Accessory Sinuses of the Nose, Laryngoscope **17**:445-450, 1907.

5. Coakley, C. G.: A Case of Tuberculosis of the Antrum of Highmore, New York Univ. Bull. M. Sc. **2**:121, 1902.

6. Kechwick, J.: Antral Empyema of Tuberculous Origin, Brit. J. Dent. Sc. **44**:433, 1895.

7. Footnote deleted.

8. Dworetzky, J. P.: Artificial Pneumothorax in the Treatment of Pulmonary Tuberculosis and Its Effects on the Larynx, Ann. Otol., Rhin. & Laryng. **35**:42-56 (March) 1936; Laryngeal Pulmonary Tuberculosis, Am. Rev. Tuberc. **31**:443-452 (April) 1935.

9. Cooper, A. T., and Benson, O. O.: A Study of the Laryngeal Effects of Collapse Therapy in Laryngo-Pulmonary Tuberculosis, Am. Rev. Tuberc. **25**: 186-196 (Feb.) 1932.

10. McConkey, M.: Personal communication to the author; Cod Liver Oil and Tomato Juice in the Prophylaxis of Intestinal Tuberculosis, Tr. Am. Clin. & Climat. A. **55**:73, 1939.

As this disease is insidious in development—frequently without symptoms—periodic inspection of the larynx in cases of pulmonary phthisis should be the rule. Frank symptoms, as stated by Fetterolf and others,[11] occur only with active disease. Pain or soreness may be present in the early stages, but both these manifestations are more often noted after the laryngeal lesion has progressed beyond the minimal stage. Hoarseness occurs only when the vocal cords are involved or if their approximation is hindered by a pathologic condition in the posterior commissure. Often the voice remains good, with the vocal cords not touched by disease, when the condition in the epiglottis, the arytenoids and the aryepiglottic folds is far advanced. Early fatigue of the voice and throat discomfort difficult to describe are worthy of notice. Pain and smarting (often referred to the ear) when the patient swallows fruit juices suggest erosion, ulceration or sometimes edema. Of course, when aphonia, dysphagia, odynophagia or dyspnea (the last-mentioned condition due to partial laryngeal stenosis unless caused by the intrathoracic disease) occurs, the diagnosis is almost self evident. Tickling cough and dryness are exasperating and troublesome in many cases. Patients with extensive ulcerative or edematous lesions often complain of inability to swallow, with regurgitation of liquids and semisolids through the nose.

Inspection of the larynx by mirror laryngoscopy enables the experienced laryngologist to determine the presence or absence of tuberculosis in most cases. Either pallor or hyperemia of the mucous membrane is only a manifestation of the patient's general condition and is not characteristic of laryngeal tuberculosis, except that unilateral congestion of a vocal cord is to be regarded with suspicion. However, infiltration, edema, erosion and ulceration can determine a diagnosis. I have gleaned little information from the roentgenograms of the larynx that I have studied, although reports by Taylor and Nathanson[12] extol the value of this method of examination for diagnosis. Pancoast[13] also has reported favorably along these lines. If the patient has had a thoracic investigation it is important to know whether pulmonary phthisis is present and whether there are tubercle bacilli in the sputum. If he has not had physical or roentgen examination of the lungs and one suspects the presence of tuberculosis but is not sure, it is invaluable to ascertain the possible presence or history of one or more of the five cardinal symp-

11. Fetterolf, G.: The Symptomatology of Tuberculosis of the Larynx, M. Rec. **75**:143-145 (Jan. 23) 1909.

12. Taylor, H. K., and Nathanson, L.: A Roentgenologic Study of Tuberculosis of the Larynx, Am. J. Roentgenol. **32**:589-607 (Nov.) 1934.

13. Pancoast, H. K.: Roentgenology of the Upper Respiratory Tract with Especial Reference to the Larynx and Adjacent Structures, J. A. M. A. **95**:1318-1321 (Nov. 1) 1930.

toms of tuberculosis as outlined by Heise and Brown,[14] namely, evidence of tubercle bacilli in the sputum, hemoptysis, pleurisy with effusion, rales in the upper half of the lung elicited by means of the stethoscope or changes characteristic of tuberculosis observed on roentgen examination. The patient should be referred to an internist or a pulmonary specialist if necessary.

The usual sites of the lesions and their pathologic appearance are vastly important. It has been my impression for many years that the initial lesion most often develops in the interarytenoid area, which, though usually concave, may be normally level or slightly convex and smooth, so that one can determine disease only by the presence of

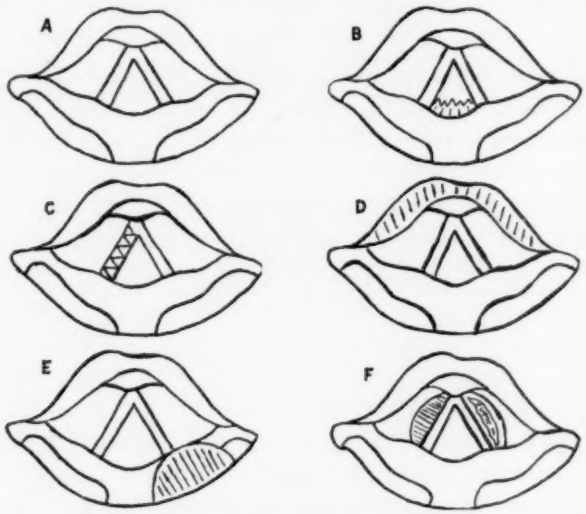

Fig. 1.—Diagrammatic sketches of the larynx, showing a normal larynx (*A*) and the site of initial lesions: (*B*) posterior commissure; (*C*) vocal cord; (*D*) epiglottis; (*E*) arytenoid cartilage, and (*F*) ventricular band or ventricle.

definite infiltration showing roughness, vegetations (tuberculomas) or erosion involving part or all of the posterior commissure; but more often the maximum change is on one side or other of the midline. However, I have been surprised to find that my statistics show that early involvement affects the vocal cords—one or both—most frequently. If the change in the cord is unilateral, it is an early helpful diagnostic sign. Hyperemia, infiltration, erosion and ulceration are the successive stages, sometimes with irregular so-called mouse-eaten edges involving one or both cords. After so many years I am still uncertain whether epiglot-

14. Heise, F. H., and Brown, L.: A Study of the Occurrence of Hemoptysis, Pleurisy, Râles, Tubercle Bacilli and X-Ray Findings in One Thousand Consecutive Cases Admitted to The Trudeau Sanatorium, Am. Rev. Tuberc. **6**:1078-1083 (Feb.) 1923.

tidian or arytenoid involvement is more common as the first manifestation of disease in the larynx, but neither is as frequent as lesions of the cord or of the commissure. In both cases the course, or advancement, is the same, namely: hyperemia, infiltration, edema, erosion and ulceration. The ventricular band or the ventricle sometimes harbors the first implantation of a tubercle. I have seen subglottic papillomatous growths twice, but the presence of tuberculosis was not proved. It must be understood that in cases of extensive, advanced laryngeal tuberculosis one may find any combination of the changes named or the entire larynx may be involved in the pathologic process. In such cases it is impossible to know which has been the initial site. There is something to the statement that there is a predilection of laryngeal disease for the side of the pulmonary involvement, but in most cases the pulmonary disease is bilateral and it is true that sometimes the larynx is mostly involved on the side opposite the more advanced pulmonary lesion. When all parts of the larynx are diseased there is usually a predominance on one side.

TABLE 1.—*Total Number of Cases of Laryngeal Tuberculosis (Encountered in Eight Years of Private Practice, 1924 to 1932)*

Tuberculous laryngitis		672
Incipient	209	
Moderately advanced	192	
Far advanced	271	

Damage to the vocal process is not classified separately, as it always involved the posterior commissure and cord.

Data on oral tuberculosis are not included in the tables except when the condition accompanied a laryngeal lesion.

Simple classification is practical. In most cases tuberculosis of the larynx is chronic, yet in some it is acute, and if in the latter cases the disease does not progress to a rapidly fatal termination it may become chronic. Less often does one see a chronic condition become acute. Intrinsic lesions are those involving the posterior commissure, the vocal cords, the ventricular bands and the ventricles. Extrinsic lesions attack the epiglottis, the aryepiglottic folds and the arytenoids. Intrinsic lesions are usually curable, whereas those of the extrinsic type offer an unfavorable or doubtful prognosis, and the massive mixed lesions are often fatal. Fetterolf's [15] central and peripheral divisions correspond to the intrinsic and extrinsic types of lesion. I classify laryngeal lesions, like pulmonary disease, into minimal, moderately advanced and far advanced, but in private practice I have had no check on the extent of the pulmonary involvement of most patients referred to me except that I know that the moderately and far advanced laryngeal lesions are more likely to occur in cases in which pulmonary disease is in a late, moderately

15. Fetterolf, G.: The Larynx in One Hundred Cases Dying of Pulmonary Tuberculosis: A Clinical Post-Mortem Study, Laryngoscope **26**:37-48 (Jan.) 1916.

advanced or far advanced stage; that is, the lesions go somewhat together. The pathologic manifestations run the gamut of vascular changes, infiltration, tubercle formation (subepithelial), granulomas (tuberculomas), edema, erosion, ulceration, perichondritis, chondritis and necrosis (or fibrosis if healing occurs)—as Spencer [16] has so aptly described.

The years of greatest incidence are the same as those for pulmonary tuberculosis, 18 to 35, and the complication is slightly more prevalent in men. I have had 3 patients in the neighborhood of 15 years of age who were girls but have not had occasion to see the condition in children. Van Poole's [17] classifications of incidence relative to race and occupation show the highest rate among Orientals and persons of the

TABLE 2.—*Age Incidence of Tuberculous Laryngitis—All Stages*

Age	Males *	Females *	Total
Under 20	7	18	25
20 to 29	103	133	236
30 to 39	149	94	243
40 to 49	91	26	117
50 to 59	36	12	48
Over 60	3	0	3
	389	283	672

* The incidence among females is seen to be slightly greater up to the age of 30. Thereafter the incidence among males predominates. Based on totals, the ratio is about 4 to 3.

TABLE 3.—*Age Incidence of Incipient Tuberculous Laryngitis*

Age	Males *	Females *	Total
Under 20	2	7	9
20 to 29	38	59	97
30 to 39	34	37	71
40 to 49	19	1	20
50 to 59	10	2	12
Over 60	0	0	0

* The incidence among females predominates up to the age of 40, then that among males predominates overwhelmingly.

laboring classes. The comparative percentage today of cases of laryngeal involvement with reference to cases of pulmonary disease suggests a revision of the figures of past years. The approximate estimate now is certainly less than 5 per cent for cases of minimal pulmonary disease, probably less than 10 per cent for cases of moderately advanced disease and not more than 20 per cent for cases in which the pulmonary infection is far advanced.

It is almost universally agreed that the laryngeal disease is always secondary to pulmonary tuberculosis. In all of the cases that I have

16. Spencer, F. R.: Laryngeal Tuberculosis, Philadelphia, J. B. Lippincott Company, 1927, p. 12.

17. Van Poole, G. McD.: Tuberculosis of the Larynx, Arch. Otolaryng. **20**: 152-161 (Aug.) 1934.

encountered the condition was such, with the possible exception of that of a woman of 55 years, seen a few days before her death one and a half years ago. Roentgenograms of the chest taken two months previously were read as normal, but the history indicated "throat trouble" of three years' duration. The involvement and destruction in the pharynx and the larynx were extensive and were typical of tuberculosis. Biopsy was refused and sputum was not available. It seems almost inconceivable that a lesion of the lung too slight to manifest itself in a roentgenogram could be the primary cause of such advanced laryngeal phthisis. Dr. Ballon [18] saw this patient and concurred in the diagnosis. He stated that he thought the laryngeal lesion was secondary to tubercle

TABLE 4.—*Age Incidence of Moderately Advanced Tubercuous Laryngitis*

Age	Males *	Females *	Total
Under 20	4	3	7
20 to 29	28	33	61
30 to 39	43	22	65
40 to 49	28	11	39
50 to 59	11	6	17
Over 60	3	0	3

* The incidence among females predominates up to the age of 30, then that among males.

TABLE 5.—*Age Incidence of Far Advanced Tuberculous Laryngitis*

Age	Males *	Females *	Total
Under 20	1	8	9
20 to 29	37	41	78
30 to 39	72	35	107
40 to 49	44	14	58
50 to 59	15	4	19
Over 60	0	0	0

* The incidence among females predominates up to the age of 30, then that among males.

deposit in the lung and that he had seen 1 or more cases in which the condition was similar.

Disagreement exists as to the manner of infection of the larynx. The consensus favors direct invasion by tubercle bacilli from sputum passing from the lungs through the larynx, the posterior part of which is frequently or constantly in contact with this infected material, the cough adding to the localized irritation. Favoring this view is the fact that most patients with laryngeal tuberculosis have evidence of tubercle bacilli in the sputum. Dworetzky reported that 91 per cent of his patients with laryngeal tuberculosis gave evidence of the presence of tubercle bacilli. McMurray,[19] in a war time postmortem study, stated to me that 65 per cent of patients with evidence of tubercle bacilli in the sputum showed

18. Ballon, D. H.: Personal communication to the author, Feb. 22, 1938.
19. McMurray, J. B.: Personal communication to the author, 1924.

some laryngeal lesion at autopsy. On the other hand, laryngeal involvement in patients in whose sputum there is no evidence of tubercle bacilli or in those who raise no sputum is not infrequent, lending support to the theory of infection by the blood stream. Gastric lavage with analysis of the contents of the stomach sometimes reveals tubercle bacilli from swallowed sputum in patients who claim not to raise any sputum. The inoculation of guinea pigs with sputum also brings out evidence that the sputum contains tubercle bacilli in some cases in which it is not otherwise observed. Myerson [20] supported the theory of a hematogenous type of infection, believing that in acutely active pulmonary tuberculosis of the caseous-pneumonic type a transient bacteremia may occur, in which bacilli are transmitted and become established in the larynx and sometimes in other organs; occasionally in miliary tuberculosis the larynx becomes rapidly and extensively involved. Greene [21] has suggested that intrinsic lesions may be caused by direct contact of sputum and that extrinsic infection is by way of the blood stream. Another possible path of transmission is by the lymphatics, but here again the theory is unproved. I agree with Dworetzky [22] that the vast majority of laryngeal lesions are surface infections from tubercle bacilli in cases of pulmonary tuberculosis of the bronchogenic type, whereas a small proportion are blood borne in cases of tuberculosis of the hematogenous type.

Differential diagnosis must exclude carcinoma and syphilis as the two diseases most likely to confuse. Syphilis of the larynx has occurred rarely in my practice (six times). The patient's history and the results of Wassermann and therapeutic tests clarify this problem. Cancer is not so easy to eliminate. Twenty patients with a malignant lesion of the larynx have come under my care for diagnosis, some already having been told that they had laryngeal tuberculosis. It is interesting to note that 1 man who had been sent home with apparently cured pulmonary tuberculosis returned to me two years later with laryngeal cancer. Thoracic roentgenograms of a patient over 45 years of age who has laryngeal involvement, which give no evidence of tuberculosis, must be regarded with suspicion. Neoplasms of the vocal cord especially are difficult to distinguish from tuberculosis. The dictum "the anterior region for a malignant lesion and the posterior region for Koch's infection" does not always hold good. Biopsy may be necessary, and the site from which the specimen is taken should be immediately cauterized,

20. Myerson, M. C.: Tuberculosis of the Larynx, Quart. Bull., Sea View Hosp. **4**:127-137 (Oct.) 1938; **4**:217-287 (Jan.); 339-366 (April) 1939.

21. Greene, J. B.: Some Aspects of Laryngeal Tuberculosis, Arch. Otolaryng. **26**:18-28 (July) 1937.

22. Dworetzky, J. P.: Modern Concepts of Laryngeal Tuberculosis, Ann. Otol., Rhin. & Laryng. **47**:481-501 (June) 1938.

regardless of the diagnosis, to prevent the spread of disease, and if microscopic examination of tissues shows changes characteristic of carcinoma immediate operation is imperative if the patient's general condition will permit. I have not encountered lupus, leprosy or actinomycosis. Laryngeal polyps and chronic nonspecific laryngitis have never presented a disturbing diagnostic problem—the former naturally not simulating tuberculosis in any manner whatsoever. Paralysis of the right or the left vocal cord may cause hoarseness, and this symptom may occur without laryngeal disease, usually due to some pressure on the recurrent laryngeal nerve in the chest, possibly from tuberculous glands or from subclavian or aortic disease.

The prognosis in cases of laryngeal tuberculosis depends greatly on the pulmonary lesion and the response of both larynx and lungs to specific treatment. The disease is no longer looked on as hopeless and the accompanying tables will show that this attitude is warranted. I do want to state that epiglottidean tuberculosis, which all these years has

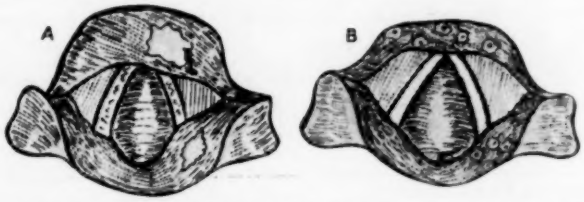

Fig. 2.—Diagrammatic sketches of the larynx (*A*) with the disease in the active stage and (*B*) with the lesion healed. *B* shows healed pitting and ignipuncture. Mr. A., aged 55, came under my care April 8, 1932. Inspection of the larynx showed thickening of the left central third of the epiglottis with grayish erosion. There was a punched-out ulceration on the left arytenoid cartilage; both vocal cords were red and thickened (infiltrated), and there was partial aphonia but no pain. The patient had bilateral advanced fibrotic pulmonary tuberculosis. Electrocauterization was performed every two weeks, and by November 11—a period of approximately seven months—the laryngeal lesion was completely healed, with satisfactory return of the voice. After a few more months of treatment for his pulmonary lesion the man left for home apparently cured and has remained well since.

been given a gloomy outlook in many texts and articles, has yielded so well to therapy that it deserves a more optimistic view. That was well demonstrated at the last school session, when my associates and I showed a goodly number of epiglottic lesions, all of which had healed or were improving satisfactorily. Occasionally they do, of course, end fatally, especially when they advance to other parts of the larynx. Laryngeal lesions in pregnancy are said to offer a bad prognosis, but I have had

no experience with such cases. Greene's [23] excellent article on prognosis shows how much more favorably laryngeal tuberculosis may be regarded today and proves the effectiveness of the newer methods of treatment in recent years. He stated that oral or aural tuberculosis in conjunction with laryngeal tuberculosis adds to the gravity, and I believe this to be

TABLE 6.—*Data on Two Hundred and Nine Cases of Incipient Tuberculosis of the Larynx*

Type of Lesion	Males		Females		Total
	Improved *	Unimproved †	Improved *	Unimproved †	
Intrinsic	96	2	95	3	196
Extrinsic	4	0	5	0	9
Mixed	1	0	3	0	4

* The word "improved" is used in this and the subsequent tables to mean improved or healed.

† The word "unimproved" is used in this and the subsequent tables to mean unimproved or dead.

TABLE 7.—*Data on Cases of Moderately Advanced Tuberculous Laryngitis*

Type of Lesion	Males		Females		Total
	Improved *	Unimproved †	Improved *	Unimproved †	
Intrinsic	76	8	50	5	148
Mixed	21	2	8	2	33
Extrinsic	10	0	1	0	11

* † See footnotes, table 6.

TABLE 8.—*Data on Cases of Far Advanced Tuberculous Laryngitis*

Type of Lesion	Males		Females		Total
	Improved *	Unimproved †	Improved *	Unimproved †	
Mixed	43	113	22	58	236
Extrinsic	3	5	1	6	15
Intrinsic	3	1	7	9	20

* † See footnotes, table 6.

true. Recurrences after complete healing are rare, but I have seen them occasionally, mostly in patients who have had an exacerbation of pulmonary disease. Examinations of the blood, including red cell, white cell and differential counts, hemograms and sedimentation rates, are a distinct aid in prognosis, and I have found this to be so during the years that I have observed such investigations at our clinics.

My early years of experience in the pulmonary disease service of a nationally known sanatorium have been of distinct help to me in the treat-

23. Greene, J. B.: Prognosis in Laryngeal Tuberculosis, Arch. Otolaryng. **26**: 18-28 (July) 1937.

ment of laryngeal tuberculosis, because it is invaluable to have a comprehensive understanding of the pulmonary disease and its treatment according to the rules of Baldwin,[24] Brown [25] and others and to realize the importance of well regulated scientific medical and surgical care of the patient with the latter disease. Cooperation with the lung specialist, the roentgenologist and, if needs be, the thoracic surgeon, is the key to successful handling of the laryngeal problem. It is important to eliminate foci of infection in the teeth, tonsils and sinuses. I firmly believe that local laryngeal treatment is of inestimable value and is the means of saving many lives. Myerson [26] expressed doubt of the curative effect of any measures applied directly to the larynx, believing that the healing power of the tissues is of primary importance.

It is the contention of most laryngologists dealing with this problem that electrocautery is the most important of all measures of attack in eradicating a tubercle deposit in the larynx. Since its introduction by

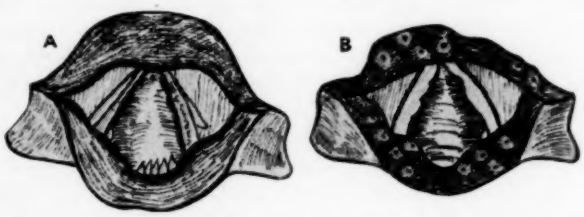

Fig. 3.—Diagrammatic sketches of the larynx (*A*) with the disease in the active stage and (*B*) with the lesion healed. *B* shows healed pitting from ignipuncture. Mr. B, aged 33, came under my care Nov. 17, 1925. Inspection of the larynx showed the epiglottis and both arytenoid cartilages to be swollen, edematous and pale; both vocal cords were markedly thickened (infiltrated with beginning ulceration), and there was interarytenoid thickening. There were complete aphonia and pain. The patient had bilateral advanced fibrotic pulmonary tuberculosis. He had been told by his city physician that his case was hopeless. Electrocauterization was performed at two, three and four week intervals over a period of fourteen months, with complete healing of the laryngeal lesion. After this, however, for more than six months ultraviolet radiation from a water cooled mercury vapor arc lamp was applied intralaryngeally, during which time the voice returned, at first deep and hoarse, but with some reeducation it became almost a normal bass voice, which has been useful and usable to the patient to this day. He has remained perfectly well and has been in business for several years.

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24. Baldwin, E. R.: Tuberculosis: Its Cause, Prevention and Treatment, Chicago, American Medical Association, 1914.

25. Brown, L.: General Measures in the Treatment of Laryngeal Tuberculosis, Ann. Otol., Rhin. & Laryng. **30**:904-911 (Dec.) 1921.

26. Myerson, M. C.: Tuberculosis of the Larynx, Quart. Bull., Sea View Hosp. **4**:470-478 (July) 1939.

Gruenwald [27] in Germany in 1905 such able men as Wood,[28] Fetterolf,[29] Levy,[30] Thomson,[31] Greene,[32] Spencer,[33] H. B. Brown,[34] Looper and Schneider,[35] Kemler,[36] Vandevere,[37] Schuster,[38] and Muskat [39] have made extensive use of this facility with increasingly good results. In all the years I have practiced, this agent has been the most potent weapon against a hostile foe, has produced healing in many cases and has served to alleviate much pain and suffering, so that I [40] believe its efficacy is unquestioned. Many patients in whom the condition was far advanced and had remained unimproved through months of watchful waiting have been healed dramatically and quickly by means of galvanocauterization. I have rarely had to resort to the injection of alcohol into the superior laryngeal nerve. Schuster [41] lauded this method for the relief of pain

27. Gruenwald, L.: Some Observations of the Treatment of Laryngeal Tuberculosis, Laryngoscopy **20**:637-644, 1905; cited by Greene.[21]

28. Wood, G. B.: (*a*) The Actual Cautery in the Treatment of Localized Tuberculous Lesions, Ann. Otol., Rhin. & Laryng. **20**:531-535 (Sept.) 1911; (*b*) The Use of the Electric Cautery in Laryngeal Tuberculosis, Am. J. M. Sc. **163**:854-858 (June) 1922; (*c*) Laryngeal Tuberculosis, Arch. Otolaryng. **21**:210-222 (Feb.) 1935; (*d*) Laryngeal Tuberculosis, ibid. **23**:236-244 (Feb.) 1936; (*e*) Tuberculosis of the Upper Part of the Respiratory Tract, ibid. **27**:476-487 (April) 1938.

29. Fetterolf, G.: The Use of the Galvano Cautery in the Treatment of Tuberculosis of the Larynx, Laryngoscope **34**:290-299 (April) 1924.

30. Levy, R.: Laryngeal Tuberculosis, J. A. M. A. **60**:1518-1523 (May 17) 1913.

31. Thomson, St. C.: Tuberculosis of the Larynx: Ten Years' Experience in a Sanatorium, London, His Majesty's Stationery Office, 1924.

32. Greene, J. B.: The Electrocautery in the Treatment of Laryngeal Tuberculosis, Ann. Otol., Rhin. & Laryng. **33**:193-197 (March) 1924.

33. Spencer, F. R.: Laryngeal Tuberculosis, Philadelphia, J. B. Lippincott Company, 1927, p. 12.

34. Brown, H. B.: Laryngeal Tuberculosis, Dis. of Chest **1**:14-15 (March) 1935.

35. Looper, E. A., and Schneider, L. V.: Laryngeal Tuberculosis: A Study of Five Hundred Cases Treated at Maryland State Sanatorium from 1923-1928, J. A. M. A. **91**:1012-1018 (Oct. 6) 1928.

36. Kemler, J. I.: Laryngeal Tuberculosis, Arch. Phys. Therapy **15**:24-28 (Jan.) 1934.

37. Vandevere, W. E.: Laryngeal Tuberculosis and the Electric Cautery, Dis. of Chest **1**:13-17 (May) 1935.

38. Schuster, F. P.: Observations of the Larynx in the Tuberculous, Arch. Otolaryng. **24**:23-36 (Jan.) 1937.

39. Muskat, I.: Electro-Cautery in Treatment of Tuberculosis of the Larynx, Eye, Ear, Nose & Throat Monthly **13**:373-374 (May) 1937.

40. Wilson, G. E.: Comparative Value of Therapeutic Measures in Laryngeal Tuberculosis, Arch. Phys. Therapy **15**:29-32 (Jan.) 1934.

41. Schuster, F. P.: Observations of the Larynx in the Tuberculous, Arch. Otolaryng. **24**:23-36 (Jan.) 1937.

along with the use of the electrocautery. Fetterolf's [42] experiments with methylthionine chloride (methylene blue) on cadavers show how definitely one can infiltrate the nerve. Schugt [43] mentioned that instead of using the external route he injected the superior laryngeal nerve by way of the piriform sinus with a curved needle, and in some cases in which there was severe pain he found it necessary to resect the nerve. In cases in which it was necessary to relieve pain from lesions of the epiglottis or pharynx he injected the pharyngeal nerve plexus with alcohol and has treated unilateral painful laryngeal disease by immobilizing the hemilarynx with an injection of alcohol into the recurrent laryngeal nerve. Wood [28] in 1911 showed experimentally that localized tuberculous ulcers produced in the abdomens of guinea pigs could be completely healed in three weeks by electrocauterization, that new blood vessels are formed, leading to fibrosis, and that prevention of the spread of infection is accomplished by the sealing of blood and lymph vessels. Hence, today

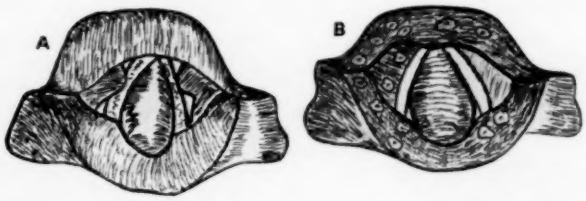

Fig. 4.—Diagrammatic sketches of the larynx (*A*) with the disease in the active stage and (*B*) with the lesion healed. *B* shows healed pitting from ignipuncture. Mr. C., aged 59, came under my care June 10, 1931. Inspection of the larynx showed the epiglottis and both arytenoid cartilages to be markedly enlarged. There was a small tuberculoma at the junction of the right vocal cord and the right arytenoid cartilage; the ventricular bands were infiltrated; the vocal cords were thickened, red and granular (infiltrated), and there were partial aphonia and pain. The patient had a far advanced bilateral pulmonary lesion. Electrocauterization was performed at two week intervals over a period of seven months, with complete healing of the laryngeal lesion. The patient continued to rest in a private sanatorium for several more months, during which time his pulmonary lesion came under complete control. He adopted Saranac Lake as his home and, being a man of means, traveled extensively for the next few years, remaining apparently well. He died in 1937 from a heart lesion.

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42. Fetterolf, G.: The Relief of Pain in Advanced Tuberculosis of the Larynx by Means of Injections of Alcohol into the Internal Laryngeal Nerve, Ann. Otol., Rhin. & Laryng. **21**:128-144, 1912.

43. Schugt, H. P.: Superior Laryngeal Nerve Block Via the Pyriform Sinus in Tuberculosis of the Larynx, Am. J. Surg. **7**:659-661 (Nov.) 1929; Tuberculosis of the Larynx: Treatment by Surgical Intervention in the Superior and Inferior Laryngeal (Recurrent) Nerve; a Report Based on Seventy-Nine Cases, Arch. Otolaryng. **4**:479-488 (Dec.) 1926; Diagnosis and Surgical Treatment of Tuberculosis of the Larynx, ibid. **21**:175-189 (Feb.) 1935.

curettage or cutting operations on tuberculous larynxes are rarely performed. Amputations of the epiglottis, as performed by Lockhard,[44] Levy[45] and others in the past, which sometimes resulted in cure but occasionally was followed by a fuminating spread of disease, is almost obsolete. Sometimes, however, the edge of the epiglottis may be snipped off and the cautery needle applied at once. Galvanocautery alone will safely heal the tuberculous epiglottis in most instances—slowly, over months of time, but without danger.

I never use electrocautery in cases in which the lesion is minimal except occasionally to destroy interarytenoid tuberculomas or vegetations interfering with approximation of the vocal cords. Consequently, my

TABLE 9.—*Data on Patients with Incipient Tuberculous Laryngitis*

	Number of Cases	Per Cent
Total number	209	
Sex:		
Male	103	47.55
Female	106	52.45
Treatment:		
Light	5	2.5
Galvanocautery	2	1.0
Result of treatment:		
Improved *	204	98
Unimproved †	5	2
Site of lesion:		
Posterior commissure	86	41.17
Vocal cord	56	26.00
Posterior commissure; vocal cord	48	23.04
Arytenoid cartilage	7	3.43
Vocal cord; B. B.	6	3.00
Vocal cord; arytenoid cartilage	2	1.00
Epiglottis	1	0.50
Epiglottis; arytenoid cartilage	1	0.50
Ventricular band; arytenoid cartilage	1	0.50
Posterior commissure; arytenoid cartilage	1	0.50
Total	209	

* † See footnotes, table 6.

percentage of cures by cautery is lowered, as I believe that in nearly all cases the lesion in an early stage will heal without cauterization. I use it fairly frequently in cases in which the condition is moderately advanced, and I resort to this modality in nearly all cases in which the disease is far advanced except the ones in which there are high fever and emaciation. Even in the latter the treatment is occasionally used to relieve pain, but in such there is always the hazard that ignipuncture may inaugurate sloughing with disintegration of tissues and denudation of cartilages. Best results are obtained if the pulmonary lesion is quiescent or only slightly active, and the better the general physical condition of the patient, the brighter the outlook for eventual recovery. Naturally, the

44. Lockhard, L. B.: Tuberculosis of the Nose and Throat, St. Louis, C. V. Mosby Company, 1913, p. 58.

45. Levy, R.: The Surgical Treatment of Laryngeal Tuberculosis, Ann. Otol., Rhin. & Laryng. **30**:912-921, 1921.

greater the number of minimal lesions submitted to cautery, the higher the percentage of patients improved or healed by the procedure. Laryngeal lesions resolve in some patients when the pulmonary disease is advancing. In fact, I have seen a few minimal laryngeal lesions healing while the patients were dying of pulmonary tuberculosis. On the contrary, I have never seen the lungs improve when the lesion in the larynx was progressing unfavorably.

Galvanocautery is an adaptable procedure and preferably should be performed by indirect or mirror laryngoscopy, especially as many

TABLE 10.—*Data on Patients with Moderately Advanced Laryngeal Tuberculosis*

	Number of Cases	Per Cent
Total number	192	
Sex:		
Male	112	58⅓
Female	80	41⅔
Result of treatment:		
Improved *	175	91⅔
Unimproved †	17	8⅓
Treatment:		
Galvanocautery	17	8.4
Light	8	4.0
Site of lesion:		
Posterior commissure; vocal cord	113	59.37
Vocal cord	14	7.2
Posterior commissure; vocal cord; ventricular band	12	6.25
Posterior commissure; vocal cord; arytenoid cartilage	9	4.68
Vocal cord; arytenoid cartilage	7	3.66
Posterior commissure; epiglottis	1	0.5
Vocal cord; ventricle palate	3	1.5
Epiglottis	6	3.1
Posterior commissure; epiglottis	3	1.5
Vocal cord; ventricular band; arytenoid cartilage	2	1.0
Posterior commissure	3	1.5
Vocal cord; arytenoid cartilage; epiglottis	2	1.0
Posterior commissure; ventricular band; arytenoid cartilage	2	1.0
Vocal cord; ventricular band	2	1.0
Posterior commissure; ventricular band; epiglottis	1	0.5
Vocal cord; arytenoid cartilage	1	0.5
Arytenoid cartilage	1	0.5
Posterior commissure; vocal cord; ventricular band; arytenoid cartilage	1	0.5
Posterior commissure; palate	1	0.5
Arytenoid cartilage; epiglottis	4	2.0
Posterior commissure; arytenoid cartilage	4	2.0
Total	192	

* † See footnotes, table 6.

patients must have it done at the bedside. Direct laryngoscopy is too formidable a procedure for use in the treatment of debilitated patients, especially as it has to be repeated and is too painful for their sore larynxes. To simplify the operation I use only sharp-pointed electrodes and have discarded the blunt ones, as the former are efficient for puncturing infiltrated or edematous areas, for searing ulcers or for eradicating tuberculomas—occasionally I even use them for cutting away the small vegetations at their bases in the posterior commissure. It is rarely necessary to cauterize vocal cords. After thorough cocainization, four to eight punctures, about ⅛ inch (0.32 cm.) apart and

TABLE 11.—*Data on Patients with Far Advanced Laryngeal Tuberculosis*

	Number of Cases	Per Cent
Total number	271	
Sex:		
Male	177	65.31½
Female	94	34.68½
Result of treatment:		
Improved *	81	29.93
Unimproved †	190	70.07
Treatment:		
Galvanocautery	172	63.46½
Light	20	7.38
Site of lesion:		
Vocal cord; aryepiglottic fold	72	26.20
Vocal cord; ventricular band; posterior commissure; aryepiglottic fold	24	8.86
Vocal cord; ventricular band; arytenoid cartilage	21	7.76
Posterior commissure; vocal cord; aryepiglottic fold	40	14.40
Posterior commissure; vocal cord; ventricular band	13	4.80
Posterior commissure; vocal cord; ventricular band; arytenoid cartilage	22	8.12
Vocal cord; ventricular band; arytenoid cartilage; epiglottis	17	6.33
Arytenoid cartilage; epiglottis	10	3.65
Vocal cord; posterior commissure; arytenoid cartilage	10	3.65
Vocal cord; arytenoid cartilage	8	2.95
Posterior commissure; epiglottis	2	0.36
Posterior commissure; vocal cord; ventricular palate	1	0.36
Vocal cord	1	0.36
Posterior commissure; vocal cord	5	1.83
Posterior commissure; arytenoid cartilage	1	0.36
Vocal cord; ventricular band; posterior commissure; epiglottis	2	0.73
Epiglottis	5	1.83
Vocal cord; epiglottis	4	1.46
Posterior commissure; aryepiglottic fold	7	2.62
Posterior commissure; ventricular band; arytenoid cartilage	1	1.10
Subglottic site	1	0.36
Posterior commissure; vocal cord; epiglottis	4	2.62
Total	271	

* † See footnotes, table 6.

TABLE 12.—*Results of Treatment*

Galvanocautery Used in 191 Cases

Stage of Tuberculosis	Patients	Each of these patients had several or many cauterizations, running the total number of sittings into hundreds	Patients
Far advanced	170		
Moderately advanced	19		
Incipient	2		
Far advanced	170		
Males	115	Improved *	26
		Unimproved †	89
Females	55	Improved	18
		Unimproved	37
Moderately advanced	19		
Males	11	Improved	11
		Unimproved	0
Females	8	Improved	8
		Unimproved	0
Incipient	2		
Males	0		
Females	2	Improved	2

Light Used in 31 Cases—Multiple Treatments

Far advanced	16		
Males	12	Improved ‡	11
		Unimproved §	1
Females	4	Improved (light only)	1
		Unimproved (1 had light only; 2 others had cautery also)	3
Moderately advanced	12		
Males	6	All improved (no cautery used)	
Females	6	All improved (3 had cautery)	
Incipient	3		
Males	1	Improved (light only)	
Females	1	Improved (light only)	

* † See footnotes, table 6.
‡ For 4 improved cautery was not used; the rest had cautery.
§ Cautery was also used for the 1 unimproved.

perhaps of similar depth, are usually made into the diseased sites with the electrode at white heat, the latter condition being important in order to prevent sticking in the tissues, which sometimes results in short but alarming hemorrhages. I have made as many as twelve to fifteen punctures in massive lesions with no serious aftermath. The procedure is repeated at two, three or four week intervals over a period of three months to one year, or longer in favorable cases, as the occasion demands. One must not insert the needle deeply into muscular areas, such as the ventricular bands or the posterior commissure, as this might cause partial stenosis of the larynx, or into the cricoarytenoid joint, risking the possibility or ankylosis. Preliminary sedatives are not necessary, and severe reactions are uncommon. I have never seen edema of the larynx following this procedure.

Stenosis of the larynx from the disease itself is not common. Only three times in my years of practice has tracheotomy been necessary, this being a procedure which merely postpones death a short time—or

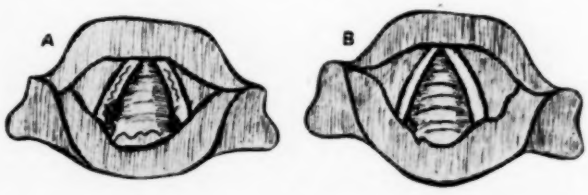

Fig. 5.—Diagrammatic sketches of the larynx (*A*) with the disease in the active stage and (*B*) with the lesion healed. *B* shows healed pitting from ignipuncture. Mrs. M., aged 31, came under my care Aug. 25, 1931. Inspection of the larynx showed the epiglottis and the arytenoid cartilages to be normal. The posterior commissure was filled with large vegetations, preventing perfect approximation of the vocal cords, which were dull gray and thickened (infiltrated), the right cord being dark red and ulcerated at the arytenoid junction. There was marked hoarseness but no pain. The patient had a moderately advanced bilateral pulmonary lesion. Electrocauterization was performed at two and three week intervals over a period of five months, during which time the tuberculoma in the arytenoid area was completely eradicated and the vocal cords improved, the latter becoming normal in appearance. This patient left Saranac Lake two months later with return of voice and apparently well and I have had no report that she has not remained so since.

such was my experience. Myerson [46] reported more extensively on its use, and in some of the patients improvement in health followed through the rest of the larynx. Healed larynxes often show distortion and deformity or partial destruction of the vocal cords, but I have known only 1 case in which the voice did not eventually return, so that I

46. Myerson, M. C.: Tuberculosis of the Larynx Requiring Tracheotomy, Arch. Otolaryng. **23**:1-14 (Jan.) 1936.

always promise patients in whom aphonia is a prominent symptom that if the larynx gets well they may be sure that they will have the use of the voice. It is true that sometimes the voice is husky for a long time and the patient needs more or less reeducation in the use of it, but it usually develops satisfactorily.

TABLE 13.—*Statistics on Cases of Laryngeal Tuberculosis Encountered in Seven Years (September 1932 to September 1939)*

Total cases		379
Minimal	156	
Moderately advanced	86	
Far advanced	137	

For treatment rest of the voice and topical application were used in all cases, alone or in conjunction with cautery or light.

TABLE 14.—*Statistics on Cases of Minimal Laryngeal Tuberculosis*

	Number of Cases	Per Cent
Total number	156	
Sex:		
Male	109	
Female	47	
Treatment and results:		
Galvanocautery		
Improved *	2	100
Unimproved †	0	
Light		
Improved	2	100
Unimproved †	0	
Vocal rest and topical applications only:		
Improved	148	97.4
Unimproved	4	2.6
Site of lesion:		
Posterior commissure	23	14.8
Vocal cord	18	11.5
Epiglottis	1	0.6
Arytenoid cartilage	1	0.6
Ventricular band	1	0.6
Vocal cords, both right and left	67	43.0
Posterior commissure; vocal cords, both right and left	36	23.1
Vocal cord, left; ventricular band, left	2	1.3
Vocal cords, both right and left; epiglottis	1	0.6
Vocal cords, both right and left; ventricular band, left	2	1.3
Vocal cord, right; posterior commissure	1	0.6
Vocal cord, left; posterior commissure	1	0.6
Vocal cords, both right and left; left arytenoid cartilage	1	0.6
Right vocal cord; right ventricular band; right arytenoid cartilage	1	0.6
Total	156	

* † See footnotes, table 6.

Light therapy has been extensively used to combat the disease under discussion and has received more than its share of favorable comment. In my hands it has given little of which to boast, and I have used both natural sunlight and artificial light of nearly all forms for local application to the larynx as well as for general bodily exposure. In one sanatorium alone there are, for universal light baths, a room equipped for treating with ultraviolet rays from air-cooled quartz mercury vapor arc

lamps, a room equipped with the carbon arc lamps, a solarium enclosed with vitaglass, which filters from the sun almost all but the ultraviolet or actinic rays, and an open air solarium for natural sunlight therapy. In my office, for local treatment I have the water-cooled quartz mercury

TABLE 15.—*Statistics on Cases of Moderately Advanced Laryngeal Tuberculosis*

	Number of Cases	Per Cent
Total number	86	
Sex:		
Male	41	
Female	45	
Treatment and results:		
Galvanocautery		
Improved *	20	83.4
Unimproved †	4	16.6
Light		
Improved	4	80.0
Unimproved	1	20.0
Vocal rest and topical applications only:		
Improved	44	77.2
Unimproved	13	22.8
Site of lesion:		
Posterior commissure	6	7.0
Vocal cord	2	2.3
Epiglottis	6	7.0
Arytenoid cartilage	5	5.8
Arytenoid cartilage; posterior commissure; vocal cord; ventricular band	1	1.2
Right arytenoid cartilage; right ventrical band	1	1.2
Right arytenoid cartilage; right vocal cord	4	4.6
Epiglottis; posterior commissure	1	1.2
Ventricular band, both right and left; vocal cords, both right and left	1	1.2
Vocal cords, both right and left; right arytenoid cartilage	1	1.2
Posterior commissure; vocal cord, both right and left	25	29.0
Vocal cords, both right and left	11	12.8
Vocal cords, both right and left; arytenoid cartilages, both right and left	2	2.3
Vocal cords, both right and left; anterior commissure	1	1.2
Posterior commissure; arytenoid cartilages, both right and left	2	2.3
Left arytenoid cartilage; left ventricular band; vocal cords, both right and left; anterior commissure	1	1.2
Right vocal cord; left ventricular band	1	1.2
Vocal cords, both right and left; posterior commissure; arytenoid cartilage	1	1.2
Vocal cords, both right and left; posterior commissure; left ventricular band	3	3.5
Left vocal cord; left ventricular band; posterior commissure	2	2.3
Left vocal cord; left arytenoid cartilage	1	1.2
Epiglottis; arytenoid cartilages, both right and left	1	1.2
Vocal cords, both right and left; ventricular band, both right and left; posterior commissure	3	3.5
Right vocal cord; posterior commissure	1	1.2
Right epiglottis; right vocal cord	1	1.2
Aryepiglottic folds, both right and left; vocal cords, both right and left	2	2.3
Total	86	

* † See footnotes, table 6.

vapor arc lamp and the quartz mercury vapor glow lamp (cold quartz lamp); the latter, being portable, can be taken anywhere to the bedside. The fatigue from trips to physicians' offices for such treatments may do harm to severely ill patients that cannot be compensated by any advantages received from treatment. I have also had experience with the Verba (or Sorgo) laryngoscope for reflecting the sun's rays into the larynx. All devices of this type are little used now. I was interested

TABLE 16.—*Statistics on Cases of Far Advanced Laryngeal Tuberculosis*

	Number	Per Cent
Total number of cases	137	
Sex:		
Male	94	
Female	43	
Treatment and results:		
Galvanocautery		
Improved *	32	30.0
Unimproved †	75	70.0
Light		
Improved	0	
Unimproved	0	
Vocal rest and topical applications only:		
Improved	4	13.3
Unimproved	26	86.7
Site of lesion:		
Epiglottis	9	6.6
Epiglottis; both right and left arytenoid cartilages; both right and left vocal cords; pharynx	11	8.0
Both right and left vocal cords; both right and left arytenoid cartilages; posterior commissure	4	2.9
Both right and left vocal cords; both right and left arytenoid cartilages; right ventricular band; posterior commissure	3	2.2
Both right and left vocal cords, both right and left arytenoid cartilages; both right and left ventricular bands; epiglottis	6	4.4
Epiglottis; both right and left arytenoid cartilages; both right and left vocal cords; right tonsil; right palate; right uvula	2	1.5
Epiglottis; both right and left arytenoid cartilages; both right and left vocal cords; posterior commissure	32	23.4
Left vocal cord; left ventricular band, left arytenoid cartilage; posterior commissure	6	4.4
Both right and left vocal cords; posterior commissure	6	4.4
Both right and left vocal cords; both right and left arytenoid cartilages; both right and left aryepiglottic folds; epiglottis; both right and left ventricular bands	5	3.6
Epiglottis; left arytenoid cartilage	1	0.7
Right ventricular band; pharynx	1	0.7
Both right and left arytenoid cartilages; both right and left vocal cords; both right and left ventricular bands; posterior commissure	10	7.2
Both right and left vocal cords; both right and left epiglottis	2	1.5
Right vocal cord; right ventricular band	1	0.7
Both right and left vocal cords; both right and left ventricular bands; posterior commissure	3	2.2
Epiglottis; left arytenoid cartilage; left ventricular bands; left vocal cord; posterior commissure	1	0.7
Both right and left vocal cords; both right and left arytenoid cartilages; posterior commissure; both right and left ventricular bands; right aryepiglottis fold	2	1.5
Epiglottis; both right and left arytenoid cartilages; gums	2	1.5
Epiglottis; both right and left arytenoid cartilages; both right and left vocal cords; both right and left ventricular bands; posterior commissure	3	2.2
Epiglottis; both right and left vocal cords; posterior commissure; left ventricular band; left arytenoid cartilage	1	0.7
Epiglottis; both right and left arytenoid cartilages; both right and left vocal cords; posterior commissure; pharynx	3	2.2
Epiglottis; posterior commissure; right arytenoid cartilage; both right and left vocal cords	1	0.7
Both right and left vocal cords; pharynx	1	0.7
Epiglottis; right aryepiglottic fold; posterior pillar	1	0.7
Epiglottis; both right and left vocal cords; both right and left ventricular bands	3	2.2
Epiglottis; posterior commissure; both right and left vocal cords	1	0.7
Both right and left vocal cords; both right and left ventricular bands; posterior commissure; right arytenoid cartilage	1	0.7
Epiglottis; both right and left arytenoid cartilages; posterior commissure; pharynx; uvula; palate	1	0.7
Epiglottis; both right and left arytenoid cartilages; both right and left vocal cords	11	8.0
Epiglottis; right arytenoid cartilage; left ventricle; posterior commissure	1	0.7
Epiglottis; right aryepiglottis fold; both right and left arytenoid cartilages; both right and left ventricular bands; both right and left vocal cords; posterior commissure	1	0.7
Both right and left vocal cords; both right and left ventricular bands; left arytenoid cartilage; posterior commissure	1	0.7
Total	137	

* † See footnotes, table 6.

in the 1928 report from the Finsen Institute (Strandberg [47]), in which universal light baths of natural or artificial sunlight were advocated and local applications were rejected as of little value mainly because of the impracticality of use and the loss of intensity of the light. Miller [48] reported encouraging results from the use of the Goerz-Wessely air-cooled carbon arc lamp, an importation from the Hajekclinic, in Vienna. Experience in timing treatments in all forms of light therapy eliminates dangers, especially those to the lungs in general bodily exposure. Local application of natural sunlight to the larynx and universal bodily exposure have been used in sanatoriums and health resorts in the southwestern part of the United States and elsewhere, but no accurate statistics are available.

I am convinced that rest of the voice is an adjuvant in treatment, although Kemler [49] reported that he thought it to have no merit. The use of pencil and pad are more sparing to the voice, but effortless whispering with the lips (using only residual air), taught to conscientious

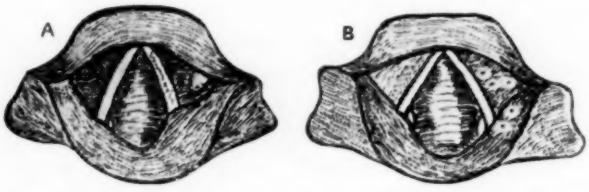

Fig. 6.—Diagrammatic sketches of the larynx (*A*) with the disease in the active stage and (*B*) with the lesion healed. *B* shows healed pitting from ignipuncture. Mrs. W., aged 50, came under my care Feb. 25, 1932. Inspection of the larynx showed the epiglottis, the right arytenoid cartilage, the right vocal cord and the right ventricular band to be normal. The left arytenoid cartilage was enlarged, the left ventricular band thickened and eroded, and the left true vocal cord red and thickened (infiltrated). There were partial aphonia and pain. The patient had an advanced bilateral pulmonary lesion. After conservative treatment over four months, during which time the patient did not improve, electrocauterization was begun and continued for more than four and one-half months, during which time the laryngeal lesion became completely healed and there was return of the voice. The patient left for home a few weeks later and, as far as I know, has remained well since.

47. Strandberg, O.: Heliotherapy and Artificial Light Treatment of Tuberculosis Particularly of Larynx, J. A. M. A. **90**:1595-1602 (May 19) 1928.

48. Miller, J. W.: Treatment of Laryngeal Tuberculosis with the Goerz-Wessely Lamp, M. J. & Rec. **123**:166-168 (Feb. 3) 1926; Artificial Sunlight in the Treatment of Laryngeal Tuberculosis, Arch. Otolaryng. **13**:326-346 (March) 1931; Wessely Radiation Apparatus in Laryngeal Tuberculosis: Further Experiments, Arch. Phys. Therapy **15**:422-425 (July) 1934.

49. Kemler, J. I.: Laryngeal Tuberculosis, Arch. Phys. Therapy **14**:24-28 (Jan.) 1934.

patients, serves a useful purpose. Granted that absolute rest of the larynx cannot be obtained, because of cough and other disturbances, pulmonary specialists depend greatly on rest in bed for the treatment of the patient whose lungs are still mobile from breathing and coughing. The point is that partial rest of the vocal cords is efficacious.

Topical application of scores of medicines has been used, but mostly with little success. Of the many drugs that I have used, none has caused healing. However, they are helpful and palliative, as they cleanse the larynx and lessen the cough. The use of chaulmoogra oil and, more recently, cod liver oil in the larynx has been praised by others, but they served no purpose in my hands and have been discarded, as have lactic and trichloroacetic acids, solution of formaldehyde U. S. P., silver solutions, a cajuput oil from Melaleuca viridiflora (gomenol) and the dyes. Guaiacol or metaphen I still use, and these should be instilled in a vegetable oil base. It has been shown that the presence of mineral oils in the larynx and trachea is dangerous, because they break up into insoluble droplets, which are not absorbed and may cause bronchopneumonia, whereas vegetable oils decompose into fatty acids and glycerin, which are absorbable. Lukens' [50] extensive and successful experience with chaulmoogra oil lends encouragement to its use, and Banyai [51] reported striking results from the local application of cod liver oil in the treatment of tuberculosis of the larynx and pharynx in various stages. Dimitry [52] stated that chaulmoogra oil deserves further trial, expressed the belief that it should not be combined with petrolatum, which may inhibit its bactericidal action, and mentioned Corper and Freed [53] as having warned against the possibility of pneumonic deposits from intratracheal instillation of chaulmoogra oil in petrolatum. Local application in the form of a spray of analgesic substances, such as cocaine or butyn sulfate, before meals in cases in which the disease has reached an advanced and painful stage and the use of hypodermic narcotics in those in which it has reached a terminal stage and there is suffering are justifiable. Tuberculin therapy is seldom used. I have had no experience with the administration of roentgen or radium treatment. Debicki's [54] results with roentgen therapy are astounding. He stated that between 1931 and 1934 he used this

50. Lukens, R. M.: Medical Treatment of Laryngeal Tuberculosis, in Jackson, C., and Coates, G. M.: The Nose, Throat and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1929, p. 885.

51. Banyai, A. L.: Cod Liver Oil for the Local Treatment of Tuberculous Pharyngitis and Laryngitis, Arch. Otolaryng. **27**:154-160 (Feb.) 1938.

52. Dimitry, T. J.: Chaulmoogra Oil in Tuberculous Laryngitis, Eye, Ear, Nose & Throat Monthly **12**:495-500 (Jan.) 1934.

53. Corper, H. J., and Freed, H. J.: Intratracheal Injection of Oils for Diagnostic and Therapeutic Purposes, J. A. M. A. **79**:1739-1743 (Nov. 18) 1922.

54. Debicki, J. J.: X-Ray Treatment of Tuberculosis of the Larynx, Eye, Ear, Nose & Throat Monthly **13**:373-374 (Nov.) 1934.

treatment in 33 cases of laryngeal tuberculosis, in which the disease was in various stages and in all of which the involvement of the lungs was far advanced (this, however, did not hinder the treatment), and he claimed complete cure in 26 cases. He emphasized the importance of exact dosage and described the technic. Terry [55] treated advanced pharyngeal tuberculosis with the high frequency current and found it of palliative value. After his personal communication I adopted the same procedure but did not elicit enough help to warrant its continuance. I have never resorted to tracheotomy or gastrostomy to rest the larynx in cases in which the disease was far advanced.

Oral tuberculosis, meaning lesions of the gums, cheeks, soft palate, uvula, tongue, tonsils, pillars and pharynx, independent of involvement of the larynx is rare in comparison to laryngeal tuberculosis, but I have encountered it fairly often. However, I have not had occasion to treat many patients with tuberculous cervical adenitis, as their cases are handled by the medical or surgical phthisiologists. Thompson [56] found

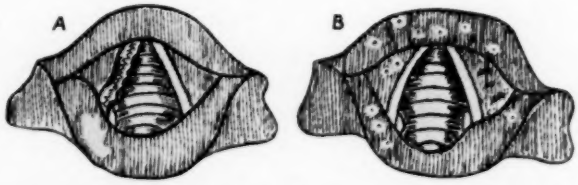

Fig. 7.—Diagrammatic sketches of the larynx (*A*) with the disease in the active stage and (*B*) with the lesion healed (before injury). *B* shows healed pitting from ignipuncture. Mrs. Z., aged 30, came under my care March 18, 1931. Inspection of the larynx showed the epiglottis to be slightly thickened and the left arytenoid cartilage, the left vocal cord and the left false cord to be normal. The right aryepiglottic fold and the right false cord were markedly swollen; the right arytenoid cartilage was thickened with erosion; the right vocal cord was dull gray, rough and thickened (infiltrated), and there were partial aphonia and pain. The patient had an advanced bilateral pulmonary lesion. Electrocauterization was performed at two week intervals over a period of five months, with apparent healing of the larynx, during which time her lungs continued to improve and the disease became quiescent, but three months later she was injured in an automobile accident, and shortly thereafter the lesions of both lungs and the larynx became reactivated, and despite cauterization and specific pulmonary treatment, this patient failed over a period of a few months and died.

the incidence of tuberculous cervical adenitis in patients with pulmonary tuberculosis the same in recent years as it was thirty years ago. While it is true that oral and laryngeal tuberculosis are infrequent companions,

55. Terry, G. H. B.: Electrosurgery in Laryngeal and Pharyngeal Tuberculosis, South. M. J. **28**:509-511 (June) 1935.

56. Thompson, B. C.: Tuberculosis of Cervical Lymph Nodes, Am. Rev. Tuberc. **37**:410-419 (April) 1938.

nevertheless they are occasionally found together. The end results of treatment of oral lesions are mostly good. These lesions are always secondary to pulmonary phthisis, and the intelligent handling of the pulmonary disease constitutes the first step in treatment. The local measures are electrocautery, light therapy and medicinal applications—of the latter, silver nitrate seems the best for this particular type of lesion. It is in the treatment of these lesions that light has been of value in my work, more than in that of any other part of the upper respiratory tract. The Kromayer lamp and the cold quartz lamp have aided from both the palliative and the curative standpoint, but here again most reliance is placed on galvanocautery, which must be used cautiously and guardedly and not too frequently. Isolated ulcers in the pharynx will heal, but pharyngeal involvement extending upward from tuberculous disease in the larynx is hopeless. Smears and cultures are unreliable for diagnosis because of the possible contamination from tubercle bacilli in the sputum. Biopsy, followed by cauterization of the wound, may be necessary to determine the diagnosis, but in many cases the experienced laryngologist can make his decision from inspection alone. One must rule out syphilis, malignant growth and Vincent's angina by the approved laboratory tests. Martin and Koepf's [57] 5 patients with tuberculosis of the gums and cheeks were all members of the male sex. The authors stated this to be the usual experience. Most of mine were males, but I have observed the condition in a few females. A most interesting experience in this connection concerned a man 47 years of age, who came to me a year and a half ago with extensive tuberculous ulceration involving the gingival region, right and left, anterior and posterior, of the lower incisors and canines and extending to the mucosa overlying the sublingual gland. This man had fairly extensive pulmonary tuberculosis of fibrotic type, and one tuberculous kidney had been removed. Complete laryngectomy had been performed two years previously, because of malignant disease—there could have been no question of the diagnosis, as the operation was performed by one of the most eminent laryngologists. I treated the oral condition a few times with quartz mercury vapor glow lamp but depended mostly on galvanocautery for results. The progress at first was slow and somewhat discouraging, but after six weeks the patient began to improve and left Saranac Lake at the end of five months completely healed. I believe it is important to bear in mind that in this case the pulmonary lesion was of the fibrotic type, thereby showing the ability of the tissues to heal, and one has the right to assume that such inherent resistance and healing power may have been present in the mucous membranes of the patient's gums.

57. Martin, C. G., and Koepf, D. W.: Tuberculosis of the Gums and Cheeks. Am. Rev. Tuberc. **37**:381-383 (April) 1938.

Tuberculous otitis media is considered secondary to pulmonary tuberculosis. All of the cases that I have encountered have been those of adult patients with definitely diagnosed pulmonary disease. Some had laryngeal lesions, and in a few the aural complication followed thoracoplasty. In 2 such cases, both those of females, the opposite ear had been deaf from childhood because of otitis media following measles or scarlet fever. Such cases, of course, are tragic. The total number of cases of otitis media due to Koch's disease that I have encountered must be large and will be reported at some future time. In every one the affliction has come on slowly with little or no pain, but in some there has been a dull ache. I have never encountered this disease as acute and painful, although such has been reported by Perkins.[58] In a few long-standing cases in which there was secondary infection and unsatisfactory drainage, pain has become troublesome. One must be on his guard in making a diagnosis, because acute pyogenic aural abscesses may occur in tuberculous patients. Again, in this complication (tuberculous otitis media) the pathway of

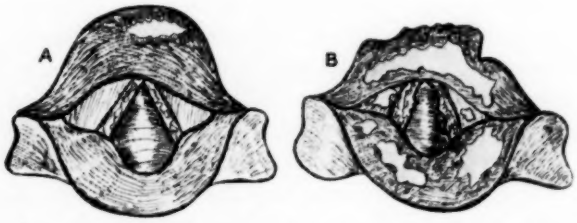

Fig. 8.—Diagrammatic sketches of the larynx with the disease active. *B* shows the extension of the lesion, the sloughing of tissues and the denudation of cartilages. Mr. P. B., aged 23, came under my care Jan. 17, 1932, with a high temperature (103 F.) and emaciation. Inspection of the larynx showed the epiglottis and both arytenoid cartilages to be markedly infiltrated and edematous, with grayish erosion on the epiglottis at the central third of the laryngeal surface. The vocal cords were dull gray and thickened (infiltrated). The patient had advanced bilateral pulmonary disease of the caseous-pneumonic type, partial aphonia and dysphagia. The diseased laryngeal tissue was cauterized on January 19, two days after arrival, with marked relief of symptoms. It was necessary to repeat the cauterization on Jan. 28, from which procedure the patient received no relief whatsoever. He continued to fail rapidly, with high fever, rapid emaciation and marked sloughing and destruction of the laryngeal structures, and on February 20, after one month's stay, he was taken to his home in a city at some distance, where he died shortly thereafter. This outcome demonstrates the inadequacy of any local laryngeal treatment in cases of this type.

infection is undetermined—whether it is through the blood or the lymph or from bacilli-laden sputum forced by cough through the eustachian tube.

58. Perkins, C. E.: Manual of Otology, Philadelphia, Lea & Febiger, 1916, p. 410.

The symptoms are few—usually stuffiness and discomfort in the ear followed by some impairment of hearing and tinnitus. By the time the patient presents himself to the otologist the tympanic membrane is thickened and appears dull dark red and the usual sharp landmarks are rounded out or less distinct. In the majority of cases, within a shorter or longer period (one day to several weeks) the drum ruptures spontaneously and painlessly. First appears a thin watery discharge, which soon thickens and becomes somewhat purulent. In most cases it is not profuse, but it continues indefinitely. Multiple perforations have been described in many texts, but mostly I have seen a single perforation, which is usually in the anterior inferior quadrant but may occur in any part of the tympanic membrane. It usually enlarges with the loss of tympanic membrane tissue. Sometimes I have seen two perforations, which later coalesced, but seldom more. In the rare cases in which the otitis does not respond to treatment bony necrosis may occur, and marginal perforations are suggestive of it; if this happens the ossicles may be destroyed and the mastoid or labyrinth may be invaded. Sequestrums have been described, but I have never seen this complication. I have had occasion to operate on but one tuberculous mastoid. I have encountered 2 cases in which facial paralysis occurred as a single complication and have had some in which there was cholesteatoma. Extension may produce meningitis or brain abscess, but I have never met with these serious metastases. Tubercle bacilli found in smears of aural pus or in cultures of it on Petroff's [59] medium or by inoculation of guinea pigs would not absolutely prove the diagnosis, as bacilli may be forced through the eustachian tube with sputum. Biopsy of granulations of the middle ear, if these are accessible, is conclusive if positive, but the procedure has certain hazards. Pathologically the diseased parts show round cell infiltration, giant cells, tubercles and granulations, and necrosis or fibrosis, depending on whether the disease progresses or retrogresses.

The prognosis is usually good, and in most cases there is little disturbing effect on the general health, nor is there much evil influence on the pulmonary condition. I have never seen tuberculous otitis media the cause of death. In treatment the building up of the general health and making sure that the pulmonary lesion is receiving proper specific therapy help to overcome the activity of the aural lesion. The most menacing end result is the marked loss of hearing in the diseased ear. Involvement is almost always unilateral, but I have encountered cases in which it was bilateral, that is, both middle ears were tuberculous. After spontaneous perforation the local treatment is practically the same as that for chronic purulent otitis media of pyogenic origin, that is,

59. Petroff, S. A.: Variations of the Tubercle Bacillus When Cultivated on Potato Media, Nat. A. Study & Prev. Tuberc. Tr. **9**:341-343, 1913.

cleansing and the use of antiseptic solutions, such as boric acid, mercury bichloride and alcohol drops or Calot's solution. I have not used zinc ionization in any of these cases. Usually the discharge gradually ceases over a three to six months' period or longer, leaving a dry ear with much scar tissue and a marked impairment of hearing, which is nearly always permanent, the hearing improving only slightly over the ensuing years. In some cases the condition does not yield to treatment, the ear discharges indefinitely and the situation is always dangerous from the standpoint of possible intracranial extension. Tuberculins or vaccines are of no value. Ultraviolet rays have been of no service whatsoever in my hands in these cases. Cox and Dwyer,[60] in reporting 25 cases of tuberculous otitis media, said that tuberculin had been found effective in treatment. They stated that 15 per cent of all discharging ears in childhood were tuberculous and that often the organism was bovine in type. In children the disease is frequently extensive and destructive to the temporal bone,

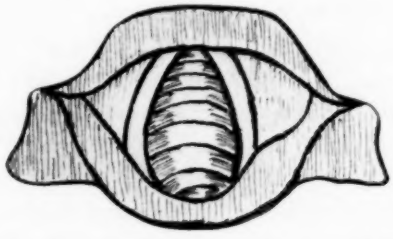

Fig. 9.—Diagrammatic sketch of a normal larynx.

whereas in adults it is usually limited to the middle ear, and it is true that 1 patient on whom I operated for mastoid involvement was a boy of 8, whereas all my other patients have been adults. Miller's[61] report on aural tuberculosis contains an excellent review of the literature. Strandberg[62] advised the use of universal light baths in preference to local applications of light to the ear. Weinstein and Bendove[63] claimed excellent results in the treatment of tuberculous otitis media by the local application of ultraviolet rays, and they did not use general irradiation for fear of harming the pulmonary lesions. I have found no definite advantage in the use of myringotomy before spontaneous perforation,

60. Cox, G. H., and Dwyer, J. G.: Tuberculosis of the Middle Ear, Arch. Otolaryng. **9**:414-421 (April) 1929.

61. Miller, J.: Aural Tuberculosis, Arch. Otolaryng. **20**:677-692 (Nov.) 1934.

62. Strandberg, O.: Heliotherapy and Artificial Light Treatment of Tuberculosis Particularly of Larynx, J. A. M. A. **90**:1595-1602 (May 19) 1928.

63. Weinstein, A. J., and Bendove, R. A.: Ultra Violet Treatment in Chronic Otitis Media in the Tuberculous, Arch. Phys. Therapy **15**:419-421 (July) 1934.

because, whether or not one resorts to incision, the drum is partially lost in many cases. In some few cases the disease resolves without perforation, and the drum is saved but the hearing is reduced. It is necessary to differentiate tuberculous otitis media from syphilis, which is rare in the middle ear, and from chronic purulent otitis media of pyogenic origin. Tuberculosis of the petrous apex has been reported recently by Grabscheid.[64]

Tuberculous lesions of the trachea and bronchi have caused widespread investigation throughout sanatoriums and hospitals only in recent years, although they were described as early as 1881 by Eppinger.[65] My work has been done in conjunction with the department of thoracic surgery. From 3 to 6 patients with lesions of this type have been subjected to bronchoscopic examination weekly for the past two years and a few at intervals previously. The accuracy of clinical diagnosis of these conditions is striking. For instance, of 21 patients referred from one sanatorium, whose condition had been diagnosed by the clinicians from signs, symptoms and roentgenograms as tuberculous tracheobronchitis, 18 gave positive bronchoscopic evidence of the disease. From other sanatoriums and more recently from the same institution the percentage has not been so high. Clinical symptoms and signs are characteristic and rather constant, although all are not present in every case, namely, wheezing, rhonchi, dyspnea (sometimes asthmatic in type), more or less convulsive cough with tenacious sputum, toxic attacks with or without fever, which are rarely followed by spread of pulmonary disease, striking variation in the daily amount of sputum which may persistently give evidence of containing tubercle bacilli not explained by the pulmonary findings. Roentgen rays disclose in some cases large or small areas of atelectasis and sometimes cavitations in collapsed or uncollapsed lungs which are not draining properly, suggesting partial stenosis of the bronchi, which may be due to the presence of tuberculous or scar tissue. There may also be some retraction and areas of emphysema. These lesions are secondary to pulmonary disease, from which they receive their infection. This is reasonable to assume, as all the patients involved have pulmonary tuberculosis but only a few have laryngeal tuberculosis, so that it is not likely that the infection is carried down from the larynx. Again arises the question of whether infection is by way of blood or lymph or sputum.

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64. Grabscheid, E.: Tuberculosis of the Petrous Apex: Report of a Case, translated by P. A. Campbell, Arch. Otolaryng. **25**:11-16 (Jan.) 1937.

65. Eppinger, H., in Klebs, E.: Handbuch der pathologischen Anatomie, Berlin, A. Hirschwald, 1880, vol. 2, pt. 1; cited, Tuberculous Tracheobronchitis, editorial, J. A. M. A. **113**:416 (July 29) 1939.

Samson [66] classified the lesions as (1) submucosal, or nonulcerative, and (2) ulcerative, whereas Myerson [67] divided them into (1) proliferative and (2) destructive. I have encountered both superficial and deep lesions of the trachea and the bronchi. The superficial ones display hyperemia, congestion, sometimes edema, infiltration and small granulations on the surface, and the deep ones manifest ulcerations penetrating almost to the cartilages, of which, however, I have not seen destruction. The lesions are usually unilateral and occur in the trachea, especially near the carina (tracheobronchial junction), in the main stem bronchi and at the openings of the lobe bronchi. Admitting the probability of the extension from below upward I have nevertheless mentioned the sites from above down, as the bronchoscopist encounters them. All the lesions that I have seen have been unilateral. If they heal with fibrosis there may result partial stenosis, and I have seen almost complete stenosis occasionally. Efforts to dilate stenosed bronchi have not been very successful but have resulted in no harm, as these procedures have been performed cautiously. I would not try to dilate where the obstruction is due to tuberculous granulations.

The agents used in treatment are high frequency current, galvanic current, ultraviolet rays and silver nitrate. Opinions differ as to the efficacy of local treatment. Naturally, healing is influenced by the activity or inactivity of the pulmonary disease and by the patient's resistance and general condition. Pneumothorax and thoracoplasty may not have a good influence on many of these lesions, and if such operative therapeutic efforts should interefere with drainage they might be harmful. Hence a definite diagnosis is important in deciding for or against the use of mechanical measures in the treatment of the pulmonary disease and also because the tracheobronchial disease itself may cause the death of the patient. Kernan [68] stated that he treated ulcerations by electrocoagulation, the application of silver nitrate or irradiation with quartz rods, that in dilating strictures he used copper ionization and that he removed tuberculomatous tissue with forceps and followed by coagulation of the base. Thus far in treatment I have used only a solution of silver nitrate, up to 20 or even 25 per cent strength (with no ill effects) and galvanocautery (see p. 32). Some patients have been treated at two to six week intervals over periods up to a year. In a few the lesions have healed, and it is possible that in some of these healing has been spon-

66. Samson, P. C.: Mucosal Tuberculosis of the Bronchi and Trachea, Dis. of Chest **4**:15-17 (June) 1938.

67. Myerson, M. C.: The Limitations of Bronchoscopy in the Treatment of Tracheo-Bronchial Tuberculosis, Ann. Otol., Rhin. & Laryng. **47**:722-734 (Sept.) 1938.

68. Kernan, J. D.: Tuberculosis of the Trachea and Main Bronchi, Ann. Oto., Rhin. & Laryng. **46**:763-770 (Sept.) 1937; cited by Wood. [28e]

taneous, without help from the medicament. Nor have I had any experience with roentgen treatments. Specimens for biopsy, in cases in which examination of tissues was indicated, have been taken, with immediate application of silver nitrate to the site and no resultant spread of the disease. The ulcers are apt to extend of their own accord, but none in my series have done so rapidly. I believe that instrumentation may possibly cause extension, so I hesitate to go beyond acutely inflamed or ulcerated areas. In a recent case a spread of the disease in the lung followed examination with the bronchoscope, but, as this was discovered by means of the fluoroscope the following day and as the patient had not been feeling well previously, though a roentgenogram had not been taken, there is some probability that the lesion was in the midst of an extension at the time she was subjected to bronchoscopic examination. At one sanatorium where roentgenograms are taken in all cases in which bronchoscopic examination is instituted, within two weeks after the procedure, collateral inflammation around the pulmonary lesion was demonstrated in some few cases, but this was shown in subsequent plates to disappear quickly.

I dislike passing the bronchoscope through tuberculous larynxes, but early involvement is not a contraindication. I was early warned against this in the case of a female patient. I waited several weeks for infiltrated vocal cords to clear, and then, as the larynx became practically normal in appearance, she decided to go elsewhere for bronchoscopic examination, because of personal affiliations. The procedure was shortly followed by the return of laryngeal involvement, and she became very ill for a few months. I have seldom encountered tuberculosis of the larynx in the cases submitted to me for bronchoscopic confirmation of the diagnosis of tracheobronchial tuberculosis. This is contrary to the report of Bugher, Littig and Culp,[69] who found laryngeal involvement in 76 per cent of their cases of tuberculous tracheobronchitis. I must say that I have been more or less at a loss to account for the fact that so few of my patients have had laryngeal deposit, as many of them were very ill and such a complication could well be expected. Nor would I employ the bronchoscope in cases in which there had been recent hemoptysis or those in which there was tuberculous pneumonia.

Laboratory data, such as sputum analyses, blood counts, hemograms and sedimentation rates, are important, as well as the results of physical examinations and roentgen studies. All my patients have had a negative Wassermann reaction. Five were proved to have carcinoma of the lung. Many have been subjected to pneumothorax, and have stood the bronchoscopic procedure well. One spontaneous pneumothorax fol-

69. Bugher, J. C.; Littig, J., and Culp, J.: Tuberculous Tracheo-Bronchitis: Its Pathogenesis, Am. J. M. Sc. **193**:515 (April) 1937.

lowed the passing of the bronchoscope, but air refills were continued by the sanatorium physicians and the pulmonary lesion healed; no tracheobronchial lesion was discovered. It is important to obtain secretions from individual bronchi for study. I should like to describe 1 case which shows the value of this procedure in the diagnostic field of tuberculosis. A woman of 38 years came to one of the sanatoriums about a year and a half ago with tuberculosis of the middle third of the right lung. There was evidence of tubercle bacilli in the sputum, and, as the roentgenograms showed no cavitation, she was treated by rest in bed alone. The pulmonary lesion cleared gradually and with satisfactory speed in ten

TABLE 17.—*Summary of Results of Treatment in Cases of Laryngeal Tuberculosis Encountered in Seven Years—September 1932 to September 1939*

	State of Laryngeal Tuberculosis						
	Minimal		Moderately Advanced		Far Advanced		
	Number	Per Cent	Number	Per Cent	Number	Per Cent	Totals
Patients							
Male	109	....	41	....	94	....	244
Female	47	....	45	....	43	....	135
Total	156	....	86	....	137	....	379
Types of treatment							
Galvanocautery							
Improved *	2	100.0	20	83.4	32	30.0	
Unimproved †	0	0	4	16.6	75	70.0	
Light							
Improved	2	100.0	4	80.0	0	0	
Unimproved	0	0	1	20.0	0	0	
Vocal rest and topical applications only							
Improved	148	97.4	44	77.2	4	13.3	
Unimproved	4	2.6	13	22.8	26	86.7	
Totals of results of all treatments							
Improved	152	97.4	68	79.1	36	26.3	
Unimproved	4	2.6	18	20.9	101	73.7	

* Improved, or healed.
† Unimproved, or dead.

months, and the sputum gave negative results. However, in the region below the hilus a circumscribed shadow remained, which was suggestive of tumor formation. Bronchoscopic examination was made with the intention of getting a piece of tissue for biopsy. This was taken from the only abnormal area seen, and, while it could not be diagnosed as a malignant growth, the experienced pathologist thought it was to be regarded with suspicion. However, on succeeding bronchoscopic examinations, secretions were obtained which were repeatedly positive for tubercle bacilli. Finally a provisional diagnosis of chronic tuberculous abscess of the apex of the lower lobe of the lung was made. Lobectomy was performed, and on removal of the lower lobe the lesion proved to be a large tuberculoma. In endoscopy I anesthetize locally with equal parts of a 10 per cent solution of cocaine and 2 per cent solution of pontocaine hydrochloride, applying this solution to the piriform sinuses and the pharynx and

instilling 1 cc. of it intratracheally. More recently I have used a 5 per cent solution of larocaine hydrochloride more profusely with satisfaction and no reactions. Tuberculous patients stand bronchoscopic procedure well, and its use is justified undoubtedly for diagnosis and treatment. It might not be amiss to subject to bronchoscopic examination all patients for whom pneumothorax, phrenicectomy or thoracoplasty is contemplated, before going ahead with these surgical measures, thereby ruling out the possibility of the presence of tuberculous tracheobronchitis, which might contraindicate the use of collapse therapy. I have found iodized poppyseed oil of some value occasionally in outlining lesions, as advocated by Goldberg [70] and others.

Recently I have devised special electrodes with which to treat tuberculous ulcers of the tracheobronchial tree by actual electrocauterization, using galvanic current. As this therapeutic measure has produced curative results in cases of laryngeal and oral tuberculosis, I believe it offers a more favorable method of attack. Neither high frequency current nor ultraviolet rays have been reported to give consistently satisfactory healing in tuberculous tracheobronchitis, and one could hardly expect such a result, as both these agents have failed in combating tuberculosis of the larynx and the mouth. I feel encouraged with the results of actual cautery to date.

SUMMARY

In this treatise I have endeavored to review present day diagnostic measures and methods of treatment of tuberculosis of the larynx and adjacent areas without attempting to invade the field of the specialist in pulmonary diseases or that of the thoracic surgeon. Most of the observations have been from personal experience over a prolonged period, and I believe they warrant the statement that worthy progress is being made in the attack on laryngeal, oral, aural and tracheobronchial tuberculosis.

70. Goldberg, B.: Endobronchial or Tracheobronchial Tuberculosis, Dis. of Chest **5**:20-21 (Oct.) 1939.

PRACTICAL POINTS IN TRANSCONCHAL RADICAL MASTOIDECTOMY

E. I. MATIS, M.D.
KAUNAS, LITHUANIA

In recent times there has been a tendency to modify the technic of the radical mastoid operation. The modifications aim at the improvement of the final results, especial attention being given to the physiologic function of the ear. Many efforts have been made to diminish operative shock (von Eicken,[1] Howarth and Bateman [2]), to establish better conditions for healing of the wound (Bockstein,[3] Lempert [4]) and to improve the hearing after the operation (Guns,[5] Tumarkin,[6] Hastings [7]). The endaural approach to the mastoid process is one of the means used in order to reach this goal.

This method, used in the nineteenth century (Kessel, Hoffman) in isolated cases, received its greatest use after the war of 1914 to 1918. It was systematically applied by Thies [8] in hundreds of cases. Certain other writers have applied it, with various modifications, in a great number of cases (Guns, Bourguet, Tumarkin, Howarth, Lempert, Bockstein).

Guns [5] modified the operative method, paying special attention to the final results. Only a few authors,[9] acting contrary to the opinion of the majority, extended the use of this method to cases of acute

1. von Eicken, K.: Zur Freilegung der Mittelohrräume vom äusseren Gehörgange aus, Ztschr. f. Hals-, Nasen- u. Ohrenh. **12**:502, 1925.

2. Howarth, W., and Bateman, G.: (*a*) The Endaural Approach to the Mastoid, Lancet **2**:1168, 1938; (*b*) Mastoid Operations, Brit. M. J. **2**:954, 1937.

3. Bockstein, T. S.: Die Radikaloperation durch den Gehörgang bei chronischen Otitiden, Monatschr. f. Ohrenh. **69**:776, 1935.

4. Lempert, J.: Einfache subkortikale Mastoidektomie, Monatschr. f. Ohrenh. **63**:1, 1929.

5. Guns, P.: La cure radicale des otorrées chroniques par le conduit et ses résultats physiologiques, Rev. de laryng. **54**:1102, 1933.

6. Tumarkin, A.: A Contribution to the Study of the Middle-Ear Suppuration with Special Reference to the Pathogeny and Treatment of Cholesteatoma, J. Laryng. & Otol. **53**:685, 1938.

7. Hastings, S.: The Operative Treatment of Chronic Mastoid Disease, J. Laryng. & Otol. **53**:246, 1938.

8. Thies, F.: Die Radicaloperation durch den äusseren Gehörgang, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:459, 1933.

9. Lempert, J., cited by Howarth and Bateman.[2a] Lempert.[4]

mastoiditis. The greater number of authors [10] pointed out the satisfactory results attained by means of the endaural operation, including the good functional effects. Notwithstanding these favorable data, specialists have not used or become familiar with the endaural methods. My personal investigations have proved that the majority of specialists do not use these methods and that many have even rejected them. Among these are specialists in former and present clinics, like those of Vienna (Neumann [11]), Stockholm (Holmgren [12]), Paris (Lemaître [12]), Berlin (Brühl [13]), Bordeaux (Portman) and many others. It must be assumed, therefore, that the endaural operative method remains in use only by persons having special interest in and aptitude for it. The fact that the endaural method has not received general appreciation, is, according to my own experience, due to the technical difficulties involved. It is generally accepted that with greater technical difficulties, a considerably narrower view of the operative field is secured. On this subject Shambaugh [14] said:

> The operation, similar to ossiculectomy with removal of the outer attic wall through the meatus, has the same disadvantages: limited visibility, limited accessibility and therefore increased danger of facial nerve injury or stapes luxation. The post-aural route has no disadvantages to compare with the increased hazard of the canal route.

By using a certain special technic it is possible to facilitate considerably and to simplify the endaural operation so that in many respects it does not differ from the classic transmastoid operation. This method gives a comparatively wide field of operation and allows one to proceed in the usual way. It is based on the following fundamental operative procedure:

1. A wide initial incision.

2. A wide subperiosteal elevation of the soft tissue of the auricle and external auditory meatus, followed by extensive laying bare of the bone, making it possible to use the mobility and elasticity of the auricle and to reach various parts of the mastoid process.

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10. In discussion on Mayer, O., and Beyer, H.: Vierzig Jahre Radikaloperation, Ztschr. f. Hals-, Nasen- u. Ohrenh. **24**:347, 1929. In discussion on papers by Engvale, Kümmel and others: ibid. **24**:447, 1929.

11. Neumann, H., in discussion on Mayer, O., and Beyer, H.: Vierzig Jahre Radikaloperation, Ztschr. f. Hals-, Nasen- u. Ohrenh. **24**:359, 1929.

12. Unpublished data.

13. Brühl, G.: Bemerkungen zur Radikaloperation des Ohres, Acta oto-laryng. **8**:475, 1926.

14. Shambaugh, G. E., Jr., in Brown, E. V. L., and others: Year Book of the Eye, Ear, Nose and Throat, Chicago, The Year Book Publishers, Inc., 1937, p. 384; cited by Tumarkin.[6]

3. The use of self-holding retractors.

4. A wide removal of the mastoid cortex.

5. The use of a special technic in removing the bone.

6. A progressive widening of the operative field in the shortest and most direct way (mastoid cortex, antrum, aditus, attic).

7. Careful attention to the tympanic membrane and ossicles.

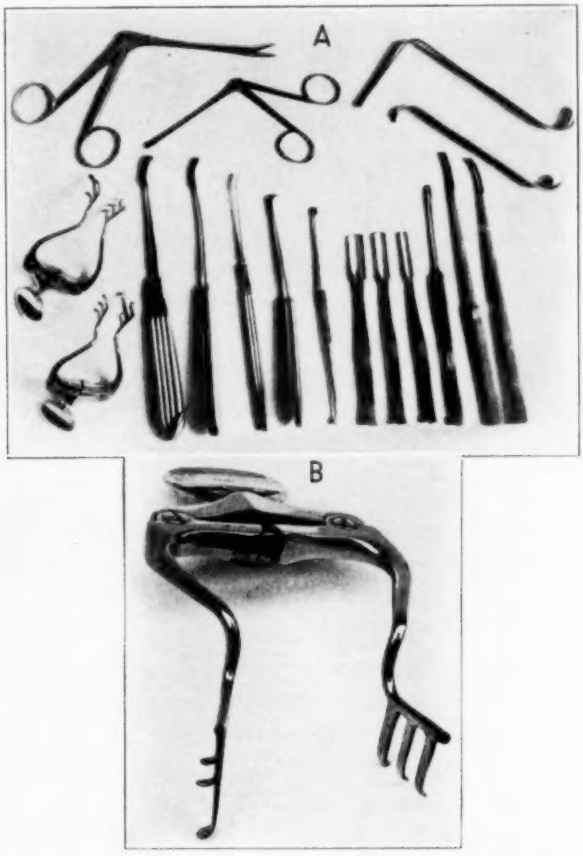

Fig. 1.—*A*, the instruments used in the transconchal radical mastoidectomy and *B*, enlarged view of the self-retaining mastoid retractor designed by the author.

TECHNIC

Instruments.—The following instruments are required (fig. 1):

1. Self-retaining mastoid retractor designed by me (fig. 1*B*). It is made in pairs, right and left, and is constructed with two blades, one terminating in two hooks; the oblong plate is rounded off at one end so that however deep it is

inserted into the external auditory meatus it cannot injure the tympanic membrane. The opposing blade terminates in a row of three hooks. It is curved to agree with the form of the auricle.

The advantages of the instrument are as follows: (*a*) it widens the operative field; (*b*) it leaves both hands of the assistant free; (*c*) it retains its position throughout the operation; (*d*) it is suitable for all sizes of meatuses, and (*e*) it automatically diminishes hemorrhage.

2. Meatus retractor.
3. Raspatories, both curved and straight.
4. Curved rugine designed by me.
5. Gouges in six widths from 2 to 10 mm.
6. Bone scoops, curets, probes and other instruments.

Administration of Anesthetic.—Local anesthesia is used, because of its many advantages; it provides the hemostasis, without which the operation would be difficult. On the other hand, although as a result of the anesthetization the soft parts swell and the external auditory meatus becomes narrower, this condition does not interfere with operation by this technic. Even when the operation is done with the patient under general anesthesia the injection of procaine hydrochloride and epinephrine hydrochloride is necessary. Five injections are given as follows (fig. 2*A*): (*1*) two in the cavum conchae, the exact site depending on the incision to be made; (*2*) one in the gap between the crest of the helix and that of the tragus; (*3*) one in the region of the apex of the mastoid process; (*4*) one for the anesthetization of the whole surface of the mastoid process.

This procedure, supplemented by filling the ear with a strong solution of cocaine and epinephrine and allowing it to remain for twenty minutes before the operation, is generally successful, except that a little tenderness often persists in the tympanic cavity.

Operation.—The initial incision is made in a semicircle through the cavity of the concha and is at least from 2 to 2.5 cm. long (fig. 2*B*). In making the incision the surgeon often stretches the auricle strongly downward and sideways and by this means causes a small wrinkle on the surface of the cavum conchae; the incision is made through this wrinkle with the knife held slantingly, point to the rear. Then, with a special curved raspatory and a rugine, the soft tissue is elevated subperiosteally. The surface of the mastoid process and the posterior and the superior wall of the meatus must be completely freed. Especially backward and downward in the direction of the apex is it necessary to free as liberally as possible (fig. 2*C*). A strip of cartilage must often be removed from the edge of the incision (fig. 2*D*). The cartilage

of the membranous posterior wall of the external auditory meatus must also be removed (fig. 3 *A*). The mastoid process is laid bare over a wide area for the purpose of orientation.

After the dermal incision is made and the periosteum elevated and separated from the posterior wall of the osseous canal and the mastoid surface, the retractor is inserted from above with the blades closed. The blade with the oblong plate is introduced between the periosteum

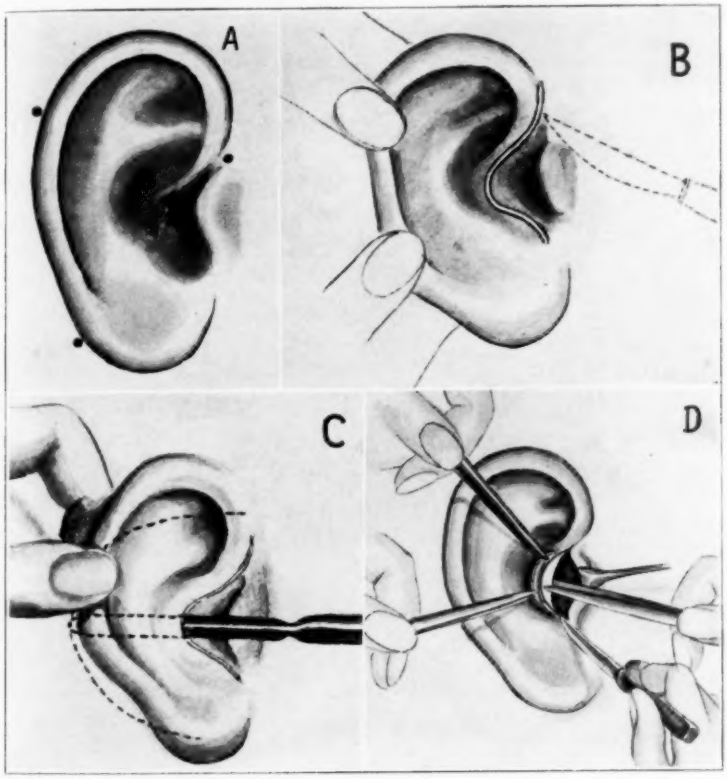

Fig. 2.—*A*, the five points where anesthesia is induced; *B*, approach by incision over the cavity of the concha; *C*, the area of the subperiosteal liberation, indicated by the dotted line, and *D*, the edge of the cartilage of the concha clipped away.

and the posterior wall of the osseous canal and must be pushed well down. The retractor is then opened, and the posterior edge of the cutaneous incision is caught up by means of the hooked blade. Next the retractor is opened wider, until the posterior edge of the incision is stretched by the hooks and the whole structure to the front is pressed against the anterior wall of the external auditory meatus by the plate blade; in this way a complete and clear view of the whole operative field is secured.

The removal of the bone is carried out from the surface to the antrum, the aim being to reach the antrum by the shortest way possible. First the bone behind the external auditory meatus is removed in the following way: A 1 cm. chisel is applied below the temporal line

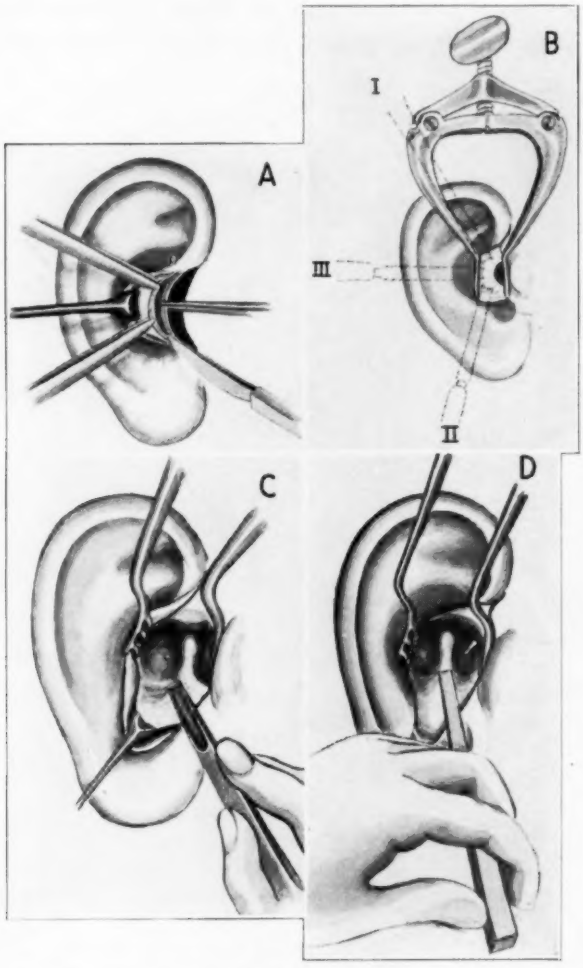

Fig. 3.—*A*, excision of the cartilage of the posterior membranous wall of the external auditory meatus; *B*, the three positions of the gouge for the removal of bone: *I*, below the linea temporalis; *II*, from the apex; *III*, parallel to the external auditory meatus; *C*, enlargement of the antrum, the operation proceeding from the mastoid apex, and *D*, the bridge carefully broken down.

and with taps of the hammer is inserted to a depth of 1 cm. The same chisel is then applied in a sloping position to the apex of the mastoid process and is directed toward the antrum. The third application is

made parallel to the external auditory meatus, about 0.5 cm. behind it (fig. 3 *B*). This procedure is repeated several times in the same order, a considerable piece of wedge-shaped bone being removed each time. In the removal of bone it is especially helpful to proceed from the apex to the antrum, as this widens the operative field in the shortest time (fig. 3 *C*). If the cutting edge of the gouge is well curved and the instrument held slantingly, there is no danger of secondary injury. In this manner the antrum can usually be opened quickly (fig. 3 *C*). In addition, a large part of the posterior wall of the external auditory meatus is removed.

Examination of the antrum shows the degree of disease of the middle ear, and on the basis of this observation a plan of the further operative procedure is made, of which the fundamental idea is to remove the diseased tissue radically and to leave the healthy parts untouched. As a rule all the diseased tissue around the antrum is removed first, and only after the mastoid cavity has been cleansed and the attic opened is the bridge broken down (fig. 3 *D*).

Up to this point the operation is easily and quickly performed, but the next act must be carried out carefully and slowly. For the purpose of preventing splinters of bone from falling inside and injuring the intratympanic structure and drum, a small piece of gauze is packed into the aditus and frequently changed. The bone is removed with a sharp chisel and in small pieces, and the utmost care must be taken to preserve intact all the normal intratympanic structures, the ossicular chain and the parts of the drum. The bridge is removed over the aditus. As this last procedure is done after the funnel-like extension of the operative field has been made, it is carried out without technical difficulty. This important part of the operation may prove hard when the attic is small or there is an anterosuperior perforation. A special technic must be used in order not to injure the drum and the ossicles. With a small labyrinth gouge, the bridge is diminished, the procedure being from the zygoma to the rear; then the inner part of the superior portion of the external osseous meatus is gradually thinned. If the bridge and the outer attic wall are divided only by a thin bony partition, this is removed by means of slight taps with the gouge, directed so as not to injure the ossicles. Of course if the hearing is bad and the clinical aspect of the case demands it, the ossicles and the drum may also be removed. I resort to this only when it is absolutely necessary.

After the newly made cavity is carefully cleaned of all splinters, the flap is cut free from the external auditory meatus; it is adapted in every endaural operation to the form and extension of the cavity on which the operation is being done. The greater part of the posterior wall of the meatus is used as a flap; it bends backward and is retained

in position by packed gauze. If the destruction of the bone is not great and the cleansed operative cavity is suitable, it may be easy to cover the bone with a Thiersch skin graft.[15] The operative cavity is filled with a small quantity of gauze. Normally, after five or six days the first change of dressing is made.

Postoperative Treatment.—Every second day, a small piece of gauze is inserted firmly into the places where there is danger of narrowing. The remainder of the cavity is lightly packed. If there is a profuse discharge, pulverization with boric acid is recommended. Granulations are cauterized with silver nitrate or lactic acid. Postoperative treatment continues until the operative cavity dries, or at least until it is certain that further improvement is impossible.

PERSONAL EXPERIENCE

The endaural operative method has been used by me in 137 cases, but technical procedure has continually been modified to keep pace with increased experience. The aim has always been to make the operative technic as simple and as easy as possible and to avoid the difficulties and disadvantages of the operative methods previously used. The first operation employed the Thies-Stacke technic, excision of a triangular flap of skin from the posterosuperior wall of the meatus, the base of the triangle being at the posterosuperior margin of the drum; extension of the operative field by means of an endaural speculum; removal of the bone, beginning from the margin of the perforation, with a small Thies gouge. Experience showed that this method was difficult and in many instances unsuitable. An examination of the numerous cases in which this operative technic was used reveals that the difficulties of the technical procedure of the endaural method of Thies and Stacke[16] are the following:

1. The greatest disadvantage of the transmeatal operation is that it is performed with monocular vision, and because of this its application is greatly complicated as the chances of judging the depth of the operative field are lost. The absence of any relief with monocular vision demands considerable more ability on the part of the operator. My investigations have proved that for this reason many do not use the transmeatal method.

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15. Shambaugh, G. E., Jr.: Primary Skin Grafts in Radical Mastoidectomy, Arch. Otolaryng. **20**:116 (July) 1934; abstracted, Ann. d'oto-laryng., 1934, p. 1295.

16. Stacke, L.: Operative Freilegung der Mittelohrräume nach Ablösung der Ohrmuschel als Radikaloperation zur Heilung veralterter chronischer Mittelohreiterungen, der Caries, der Necrose und des Cholesteatoms des Schläfenbeins, Tübingen, F. Pietzcker, 1897.

2. It is difficult to operate in the presence of a narrow external auditory meatus or a deep lying tegmen and small attic.

3. Hemorrhage greatly interferes with the operation.

4. Certain cells of the mastoid process are often difficult to reach.

5. There is a certain danger of postoperative stenosis of the entrance to the external auditory meatus, which interferes with good supervision of the radical cavity.

6. Special care of the dura mater has to be taken at the beginning of the endaural operation in order to avoid accidental injury. In several of my cases, when operating according to the Thies-Stacke method I opened the dura mater at the very beginning, and as I was using a small sharp chisel the danger of accidental injury was great.

7. Local anesthesia considerably narrows the external auditory meatus.

8. The fact that the services of an assistant must be much used also makes the transmeatal operation inconvenient. For example, the assistant has to use the hammer, hold the speculum and give other aid.

General conclusions on the Thies-Stacke technic and other methods may be summed up as follows:

Technically the endaural operation requires great skill. It is important to select suitable subjects. In the presence of cholesteatoma and a wide external auditory meatus the results are often good. In all my cases in which there was osteitis or growths of polyps, and in which the external auditory meatus and the attic were small, the final results were not so satisfactory. I have observed not only my own cases but also those in which operation was done by others, and have noted, not infrequently, that the cavity becomes narrow and consequently inconvenient for examination. The region of the aditus was often found to be grown together, and in the tympanic cavity were hidden granulations difficult to reach for the purpose of treatment and the cause of purulent discharge. In such cases the effect on the hearing was not good.

The modification of the endaural operation first described by me [17] in 1933, was aimed at simplifying operative procedure [18] and avoiding the shortcomings [19] just mentioned. To this end I had to give up the subcortical procedure and adopt the subperiosteal operation. Supporters of the endaural approach to the mastoid process (Thies, Lempert and Bockstein) assert that the fact that the cortical layer of the mastoid

17. Matis, E.: Die conchale Radikaloperation des Ohres, Ztschr. f. Hals-, Nasen- u. Ohrenh. **35**:181, 1933.

18. Matis, E.: La cure radicale par la voie conquale, opération sous-periostée, Compt. rend. Soc. franç. d'oto-rhino-laryng. **49**:118, 1935.

19. Beyer, H.: Die Operationen am Ohr, ed. 2, Leipzig, Curt Kabitzsch, 1938, p. 149.

process is left untouched is the most significant advantage of this procedure and results in better and quicker healing. This point of view is upheld by the fact that the mastoid cortex in chronic aural diseases is seldom affected when it is thick and hard. I suggest, however, that the subsequent healing of the wound after the mastoid operation depends more on the periosteal layer. This theory is borne out by physiology and by experience in general surgery, as well as by the common idea of the formation of new bone. For building of new bone it is important that the periosteum be uninjured and well nourished and especially that the blood vessels and nerves leading to it and situated under the skin remain untouched. With the subperiosteal operation, conditions which permit this are secured. The periosteum is elevated from the underlying bone in the best physiologic condition, as its connection with the overlying soft tissue remains untouched by operative action. Hence the healing of the wound and the formation of new bone come about quickly and completely, as is proved by examination of the patients operated on by the subperiosteal method. By using the subperiosteal operation, it was possible to simplify the operative technic considerably, to widen the operative field and to avoid many of the difficulties and disadvantages of the other transmeatal methods.

It was always possible to perform the operation under binocular vision; this was the most important advantage of the subperiosteal method. It rendered the technical execution of the operation considerably easier. Because the operation was carried out from the surface of the mastoid process toward its center, the most difficult and dangerous part of the procedure was after the operative field had been considerably widened and the condition of the antrum and tympanic cavity established. The subperiosteal method is better than the subcortical in that it facilitates the observation of the operative field and the removal of all the deep lying cells and branches of the cholesteatoma. A narrowing of the external auditory meatus or a swelling of the soft tissue after local anesthetization never interferes with the performance of the operation. A great advantage of the transconchal operation is the fact that the entrance to the external auditory meatus is large enough to permit observation of postoperative healing and to secure the growth of epithelium in the newly formed cavity. The ossicles and the drum can be particularly protected by this method because they are dealt with only at the end of the operation, when a good view of this region is obtainable.

The following observations on the use of the transconchal method may be noted: In my series of 137 cases, only 2 with complications were met. In 1 of these the condition was a thrombophlebitis resulting

in death, in which extraordinary circumstances played a part; in the other case the disease was perichondritis. (These 2 cases are reported later in this paper.)

All the patients with complicated mastoiditis and other persons unsuitable for operation by the endaural approach were operated on by the classic transmastoid method; for example, those with swelling or infection of the soft tissue, thrombophlebitis, meningitis, subdural or cerebral abscesses or diffuse purulent labyrinthitis. Even a suggestion of these complications demands a transmastoid operation. Only once in my practice have I used the transconchal method when afterward it appeared, contrary to expectations, that there was a cerebral abscess. That this case ended happily can be put down to the fact that the abscess was well protected by a strong membrane that had formed around it. This case has already been described in print.[20]

Of all the complications, the only ones not rendering the endaural approach inadvisable are nonpurulent and nondiffuse labyrinthitis. In cases complicated by these conditions I have tried to diminish operative shock by using the transconchal method.

In all these cases there was good healing. Ten per cent of the patients were over 60 years of age, 4 being over 70. All stood the operation well. A definite conviction has been acquired that the transconchal method is especially suitable for the aged as it can be performed with local anesthesia, and is less complicated than the transmastoid method.

The operation was performed on about 15 outpatients without meeting with any complications. The technical performance was successful in every case.

In every instance the hammer taps were given by the operator himself; the services of the assistant in this operation are not important. Indeed, when the technic described here is used the assistant can be dispensed with altogether.

On the basis of numerous demonstrations of this method, I have come to the conclusion that the whole procedure of the operation can be watched by any observer and also by the assistant himself; this goes to prove that during the work there is a wide enough field for binocular vision. The sinus and dura were exposed in many cases without any particular complications. In 1 case there was sinus bleeding which, however, it was possible to stop, and the operation was carried through to the end. Paralysis of the facial nerve was not met with in any of the cases. In my first case there was temporary paralysis of the facial nerve lasting several hours, as a result of incorrect anesthetization, but it passed away leaving no signs.

20. Matis, E. I.: Die Indikationsstellung und die technische Ausführung der Antrotomie durch das Cavum conchae und vom äusseren Gehörgang aus, Monatschr. f. Ohrenh. **70**:1230, 1936.

On the basis of the cases in which the operation on one ear was by the transmastoid method and on the other by the transconchal, I have drawn the conclusion that healing comes about more quickly after the latter type of operation. A general comparison of the cases in which operation was by the transmastoid method with those in which the transconchal method was used leads to this conclusion. It must be said, however, that individual healing of the wound must be considered. With both methods I have noted that wounds have healed quickly in some cases and slowly in others; and so the speed of healing cannot be put down to any one particular method without exception.

The functional results in most of the cases were good. This is partly explained by the fact that with the transconchal method special care was taken to protect all the structures necessary for hearing.[21] A certain part was played by the circumstance that it was the more suitable patients who were selected for the transconchal method; therefore the effect on the hearing was not so serious.

REPORT OF CASES

A series of my cases has already been described by me [22] and also demonstrated; I do not intend, therefore, to furnish here any statistical confirmation of the value of my methods. I desire only to bring forward a few unpublished reports of cases in order to illustrate certain points that have been mentioned.

It has already been stated that the transconchal method is considerably easier for aged and weak patients.

CASE 1.—C. S., a married woman aged 65, had a history of earaches and headaches for a month previous to her consultation with me; there was slight aural discharge. Examination showed the left ear to be swollen and painful to the touch. Subsequent observation proved that the swelling was due to the fact that a cholesteatoma had broken through the posterior wall of the external auditory meatus. This was a suitable case for the transconchal operation, and notwithstanding the patient's poor general condition and cardiac weakness, such an operation was decided on. On Oct. 16, 1939, with the region under local anesthesia, the transconchal operation was carried out with great ease in twenty minutes. Postoperative hearing was satisfactory.

Such voluminous cholesteatoma is easy to operate on by the transconchal method.

CASE 2.—A. V., a married woman aged 60 years, had suffered from periodic vomiting, giddiness and headaches for many years, all of which symptoms were slight. Examination showed a dry cholesteatoma of the right ear and a small

21. Young, G.; Howarth, W.; Brown, G., and Wright, A. J., in discussion on Hastings.[7]

22. Matis (footnotes 17 and 20).

attic perforation. It was difficult to decide whether all the complaints could be attributed to the ear. The probatory operation was decided on, and on Feb. 14, 1935 was performed, with the region under local anesthesia. A small antrum full of cholesteatoma was revealed. After the operation the patient went home. Healing with no discharge was complete within a month.

The good results may perhaps be put down to the fact that this was a nonsuppurating cholesteatoma.

Complications of otitis media are contraindications to all endaural operative methods. The only exceptions are noncomplicated and nonpurulent labyrinthitis following chronic suppurative otitis media. In such cases the endaural approach has the advantage of allowing more caution and producing less operative shock.

CASE 3.—S. G., a married woman aged 52 years, complained for four days of severe attacks of vomiting and giddiness which were brought on by the least movement of the head. Examination showed spontaneous nystagmus of the third degree, an attic perforation and a foul-smelling cholesteatoma. There was no tenderness or swelling of the mastoid region. Hearing was almost entirely absent. A consultation among a number of specialists resulted in the decision to wait a few days. On July 3, 1935 a transconchal operation with the region under local anesthesia was performed. The sinus was far forward, making it difficult to approach the antrum. After the antrum was opened and the cholesteatoma removed, erosion of the external semicircular canal was established. Healing was uninterrupted and good.

CASE 4.—S. K., a woman aged 27, complained of a discharge from the left ear since the age of 4 years. Six weeks previously she had had an attack of vertigo, vomiting and headaches, which had compelled her to remain in bed for a week. On examination the left ear showed chronic suppurative otitis media with posterosuperior marginal perforation, cholesteatoma and granulations. Slight spontaneous nystagmus could still be seen when the patient looked toward the right. The deafness of the left ear was almost complete. In view of the repeated labyrinthine attacks of the left ear, a transconchal radical operation on this ear was performed on May 14, 1934. A quantity of cholesteatoma was found. When the patient was examined two years later she said she had had no further attacks of vertigo and the middle ear was dry.

That the transconchal operation gives better results for hearing than the transmastoid method may be explained on the basis of the restoring force in the auditory system (Pohlman[23]), which can be better protected by the transconchal technic.

CASE 5.—J. B., a man aged 24, a student of law, had in the right ear a small superior perforation and cholesteatoma and in the left, a central perforation. A whisper was heard in the right ear, at 0.5 meter and in the left, at 1 meter. In view of the patient's career, for which good hearing is especially important, great care was taken to preserve aural function. On Nov. 4, 1934 the operation was carefully and slowly performed, lasting about one hour and forty minutes. All the intra-

23. Pohlman, A. G.: Restoring Forces in the Mechanics of Audition, Ann. Otol., Rhin. & Laryng. **48**:148, 1939.

tympanic structure was left intact, and the cholesteatoma was cautiously removed. Examination three years later revealed that a whisper was heard in the right ear at 4 meters and in the left at 0.5 meter. The patient declared that, practically, his hearing was considerably improved.

Case 6.—G. B., an unmarried woman aged 20, had superior perforation and cholesteatoma in the right ear; a whisper was heard in that ear at 0.3 meter. In the left ear there was progressive middle ear catarrh and the whisper was heard at 1.50 meters. A transconchal operation was performed on Jan. 27, 1938. Two years later examination showed that the cavity was dry and the intratympanic structure intact. The whispered voice was heard in the right ear at 2 meters and in the left at 1 meter.

In certain cases a diminution of hearing is noticed in spite of good anatomic conditions.

Case 7.—L. G., a man aged 52, had had otorrhea in the left ear since childhood; he complained of headaches, giddiness and noises in the ear. Examination showed a superior perforation and a small polyp protruding from it. Foul-smelling cholesteatoma was observed. The whispered voice was heard at 0.2 meter. On May 22, 1934 the transconchal operation was performed. Both the external auditory meatus and the attic were large; this facilitated the operation. Examination two years later revealed a large dry cavity; the intratympanic structure was intact. Conversation was audible at 1 meter.

I cannot account for the diminution of hearing in case 7; great care was exercised during the operation and postoperative treatment.

In 2 cases there were severe complications; these cases, I think, should be made public.

Case 8.—J. M., an unmarried woman aged 20 years, was of a family in which several members had signs of degenerative hereditary diseases. The patient was deaf from birth and an imbecile. She had had otorrhea in both ears since childhood. Recently her temperature had risen and she had suffered from vertigo and headaches. Examination showed foul-smelling pus in both ears. There was spontaneous nystagmus to the right side. Slight tenderness over the left mastoid process was noted. It was difficult to make an exact diagnosis, owing to the patient's mental condition. Labyrinthitis in the left ear was suspected, and it was decided to operate by the transconchal method. The operation was done on Feb. 7, 1936, and a large area of osteitis with necrosis of the bone was found. The bone was removed as far as possible and the sinus freed for approximately 2.5 cm. The same day the patient tore off the bandages, infecting the wound. Two days later she had intermittent high fever. A second operation was performed on Feb. 9, 1936 by the transmastoid method and showed extensive thrombophlebitis, reaching to the bulbus. During the operation, many infected cells were found, especially in the region between the second and the third sinus knee. As the patient's condition did not improve, a ligature was placed on the jugular vein the next day. Ten days later general sepsis and weakness occurred, and the patient died.

It may be assumed that the cause of the complications in this case was secondary infection by the touch of the patient's fingers, although it cannot be excluded that the source of infection might have been the cells

not removed at the first operation. At any rate, I have come to the conclusion that in cases in which the picture is complicated and vague, the operation should be performed by the classic method.

CASE 9.—C. S., an unmarried woman aged 23, had suffered from chronic inflammation of the ear since childhood. She had previously had furunculosis in the diseased ear. As she had complained recently of headaches it was decided to operate. The left ear showed a fetid discharge and a posterosuperior perforation with a small protruding polyp. The transconchal operation was performed on Jan. 31, 1938. The antrum was small and contained several granulations. There was no cholesteatoma. The destruction of the bone was slight. Ten days after the operation perichondritis of the auricle appeared and became progressively worse, resulting in extraordinary deformity after six weeks.

It is difficult to decide to what degree the method in case 9 was responsible for the perichondritis. Since that complication, however, I have always taken special care in the treatment of the cartilage of the auricle.

All authors who operate according to the intrameatal method confirm the statement that operative shock is less and that patients leave the hospital sooner and quickly become fit for work (Howarth, Guns, Bockstein); my experience bears this out. I give as examples 3 cases, selected at random, in which operation was done recently (1939).

CASE 10.—S. G., a 41 year old laborer engaged in heavy physical work, had chronic otitis media with a superior perforation, cholesteatoma and severe headaches on the side of the affected ear. Operation with the region under local anesthesia was performed on Oct. 3, 1939. The next day the patient was walking about the hospital halls, and a week later he was discharged. He was able to resume work within a month. Examination on Nov. 20, 1939 revealed an almost dry cavity and slight granulations in the region of the antrum.

CASE 11.—L. T., an unmarried woman aged 24, had a foul-smelling cholesteatoma and headaches, as well as occasional attacks of giddiness. Operation was performed on Nov. 14, 1939 and a large mass of cholesteatoma was removed. After six days she was able to go home. Examination five weeks later showed complete healing.

CASE 12.—J. C., a youth aged 16 years, had posterosuperior perforation and cholesteatoma. Previously he had had an acute exacerbation of a dangerous character. The operation was performed on Nov. 19, 1939, with the region under local anesthesia. There was a cholesteatomatous mass in the antrum and in the periantral region. The mastoid process was sclerotic. After ten days the patient was discharged from the hospital. Examination five weeks later showed a slight discharge from the region of the eustachian tube. Epidermization was almost complete.

SUMMARY

In order to improve the functional results in the ear and to hasten healing, the transmeatal radical mastoidectomy was proposed by some. This method, however, was often technically difficult, especially because of monocular vision. I suggest for this purpose the so-called transconchal

subperiosteal radical mastoidectomy. This can be done under binocular vision and technically is not difficult. Performed with the region under local anesthesia the procedure is as follows: After the incision has been made through the cavity of the concha, the soft tissue and the auricle are widely elevated; a self-retaining retractor is inserted and the mastoid cortex is removed. The mastoid process is chiseled from the outside to the inside, and the antrum is exposed. Then a probe is introduced; the posterior wall of the auditory meatus is removed, and a plastic flap is formed. The ossicles and the tympanic membrane are left intact as far as possible.

The results in the 137 cases in which the operation was done were satisfactory; complications occurred in only 2. Technically the operation gave rise to no difficulties.

PROSTIGMINE METHYLSULFATE IN THE TREATMENT OF DEAFNESS AND TINNITUS AURIUM

REPORT OF FIFTY-NINE CASES WITH NEGATIVE RESULTS

MATTHEW S. ERSNER, M.D.

IRVING A. RUSH, M.D.

AND

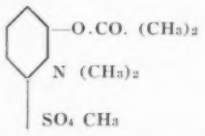
DAVID MYERS, M.D.

PHILADELPHIA

Because the use of prostigmine methylsulfate in the prevention and treatment of various disorders has been reported so favorably and further because in the recent months it has created a furor in the otologic world,[1] we began a methodical study of 59 cases, despite the fact that our past results from the employment of prostigmine in the treatment of deafness and tinnitus aurium had been disappointing.

We carefully selected our cases, making certain that in each there was freedom from sinus involvement and from obstruction of the eustachian tube. By so doing, we hoped that our results would be as gratifying as those of our predecessors but unfortunately our report, which follows, shows the contrary.

Prostigmine methylsulfate is described as "the dimethylcarbamic ester of 3-hydroxyphenyltrimethylammonium methylsulfate," with the following structural formula:



It belongs to the group of so-called autonomic drugs, being parasympatheticomimetic. The great bulk of pharmacologic evidence places the site of its action at the parasympathetic nerve terminals, i. e., at the junction of the postganglionic fibers with the end organ, where its effect is one of stimulation. It is a synthetic substance resembling physostig-

From the Department of Otology, Temple University School of Medicine.

Presented before the staff of the Department of Otology of Temple University, March 21, 1940.

1. Davis, T. C., and Rommel, J. C.: Treatment of Deafness and Contiguous Nervous Disorders with Prostigmine: Preliminary Clinical Report, Arch. Otolaryng. **29**:751-758 (May) 1939.

mine and pilocarpine [2] and acting principally on the intestinal, bronchial and bladder musculature, more rapidly and powerfully than physostigmine; hence its well known clinical use in the treatment of such conditions as postoperative paralytic ileus and atony of the bladder.[3]

In recent years much work has been done to increase knowledge of the mechanism of these drugs, such men as Goodman and Bruckner,[4] Myerson, Rinkel and Loman [5] and others [6] further clarifying present understanding. Gremels [7] in 1935, in summarizing this knowledge, said that impulses of parasympathetic nerves are transmitted by the release of acetylcholine at the nerve endings. As soon as a chemical balance is established, the action of acetylcholine disappears. He stated that it is continuously being produced, the production depending on central vagus tone, but that an esterase (cholinesterase) appears to inactivate it by hydrolysis and that this cycle of production and inactivation produces an uninterrupted activity coordinated to central vagus tone. This mechanism of potential action of acetylcholine is influenced by physostigmine and even more by prostigmine in that they inhibit action by the esterase which is responsible for the continuous destruction of the acetylcholine. The drug is, thus, essentially synergistic.[8]

At the very outset accurate hearing tests were performed on each patient; these were repeated twice weekly throughout the experiment. Since the results of all hearing tests are subjective, in some instances certain patients showed a variation of 10 decibels. In these instances we repeated the hearing tests three times at one sitting and divided the added results by 3, thus arriving at an average.

Since the results of testing hearing by means of the voice, including the whisper, are relative, we based our observations on the results

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2. Wright, H. H., and Montag, M.: A Textbook of Materia Medica, Pharmacology and Therapeutics, Philadelphia, W. B. Saunders Company, 1939.

3. Greenberg, B.; Loman, J., and Myerson, A.: J. Urol. **40**:280-284, 1938. Marden, P. A., and Williamson, E. G.: Surg., Gynec. & Obst. **69**:61-64 (July) 1939. Begg, R. C.: Brit. M. J. **2**:581 (Sept. 18) 1937.

4. Goodman, L. S., and Bruckner, W. J.: The Therapeutics of Prostigmin: Warning Concerning Its Oral Use Based on Personal Experience, J. A. M. A. **108**:965-968 (March 20) 1937.

5. Myerson, A.; Rinkel, M.; Loman, J., and Myerson, P.: J. Pharmacol. & Exper. Therap. **60**:296-311, 1937.

6. Myerson, A.; Loman, J., and Dameshek, W.: Am. J. M. Sc. **193**:198-214, 1937.

7. Gremels, H.: Arch. f. exper. Path. u. Pharmakol. **179**:360-402, 1935; cited by Myerson, Rinkel, Loman and Myerson.[5]

8. (*a*) Myerson, Loman and Dameshek.[6] (*b*) Greenberg, Loman and Myerson.[3] (*c*) Myerson, Rinkel, Loman and Myerson.[5]

obtained with the audiometer and tuning forks rather than on those with the human voice. Thus we had a definite standard by which to make comparisons.

It is a fact well known to otologists that the pitch and volume in the voice vary greatly not only in different persons but also in any given person at various times. Although with practice one can cultivate a reasonably uniform intensity and pitch of the voice, sufficient for gross estimates of hearing, it has been proved by the oscillometer that the variations even in such a controlled voice are so tremendous as to make the results hopelessly inaccurate when the voice is used in the scientific testing of hearing. The results of our comparative hearing tests are therefore based on quantitative measurements carefully graphed by calibrated audiometers.

Knowing that the physiologic action of the drug is fairly prompt but fleeting, and not cumulative, and that the administration of this drug for deafness and tinnitus is of necessity empiric, we intentionally varied the intervals between doses with the hope of determining an optimal interval.

METHOD

The drug was administered hypodermically, a dose of 1 cc. of a 1:2,000 solution (0.5 mg.) being used in each instance. The system of dosage was as follows: Twenty-five patients received daily injections; 10 patients received injection three times weekly; 14 received one injection every other day, as well as supplementary oral therapy with prostigmine bromide tablets (15 mg. each, three tablets daily), and 10 patients received injections three times weekly, supplemented with ammonium chloride (enteric) capsules.

Each patient received a minimum of thirty-five injections except those 3 patients for whom the administration of the drug was discontinued because of untoward effects.

The total number of patients was 59. In 14 of these the condition was clinically diagnosed as otosclerosis.

RESULTS

Four patients asserted that they had improved. Their statement could not be verified by the hearing tests, the audiometric readings disclosing no change. These patients said there was improvement even after administration of the drug was discontinued and that of physiologic solution of sodium chloride was substituted.

Eighteen patients said that symptomatic improvement occurred soon after the first few injections. This change was purely subjective, for the improvement in hearing could not be verified by the audiometric and tuning fork tests. After several weeks of treatment the euphoria and enthusiasm of the patients subsided, and the symptoms remained the same despite the continuation of the therapy.

Eight patients after having received fifteen injections showed an 8 to 10 decibel improvement at varying times in the course of therapy, but repeated hearing tests revealed that the improvement not only was not permanent but could not even be described as prolonged. In each case the so-called excursion of improvement was transitory.[9]

Three patients had attacks of acute angina pectoris, so that it was necessary to discontinue the administration of prostigmine. Atropine had to be administered in order to overcome the anginoid pain.

The remaining 26 patients showed no so-called improvement in the tinnitus aurium or hearing either during or after the course of therapy.

COMMENT

Prostigmine was not employed in cases of acute deafness and tinnitus aurium. We know from past experiences that not only did the acute forms respond to routine care and treatment, but in many instances they disappeared spontaneously. Therefore we did not include any such cases in this series.

The different types of deafness treated in this study were: conductive deafness in 10 patients; nerve deafness in 10; otosclerosis in 14, and deafness of mixed type in 25. Since 65 per cent of these 59 patients were middle aged, many of them had diminished bone conduction with a loss in the high as well as the low frequencies.

In middle-aged persons various geriatric changes occur besides those pertaining to the ear, the most important of these involving the cardiovascular system. When there is definite or probable cardiovascular involvement or idiosyncratic hypersensitivity to the drug, the administration of prostigmine is not without its untoward effects.[4] In 3 of the patients in this series, after the administration of the drug, acute anginoid symptoms developed, so severe and alarming that emergency treatment with atropine was required and the prostigmine therapy had to be abandoned.

In patients with high or low blood pressure tinnitus is a common occurrence. The variable pharmacologic effect of prostigmine on blood pressure notwithstanding, none of the patients with high blood pressure in this series showed any improvement in the tinnitus aurium. A few of the patients with low blood pressure were among those showing a fleeting and questionable improvement in the tinnitus.

There are physiologists and pharmacologists who believe that the administration of prostigmine may, by an indirect effect on the adrenal

9. We have observed that when patients are tested during the early part of the day the audiometric chart may show a slight improvement in hearing. On the other hand, when these same patients are tested after a hard day's work, both physical and mental, the audiometric chart frequently shows a drop in hearing.

glands, result in the secretion of epinephrine, producing a temporary elevation of the blood pressure. This, possibly, might explain the so-called temporary improvement in some cases. If this is so (and we do not know that it is), it is our opinion that these same patients would probably show a temporary improvement if they were given such sympathetic drugs as ephedrine or epinephrine instead of prostigmine. In fact such has proved, on trial, to be the case.

One should not assume, as has been done, that since prostigmine indirectly influences smooth muscle by way of the parasympathetic nerve endings, its mode of action in cases of tinnitus aurium might be due to such an indirect action on the stapedius and tensor tympani muscles, or on one or the other of these muscles since they, regardless of whether one considers them primarily as voluntary or involuntary, are histologically striated muscle. Conversely, if, cognizant of that fact, one presumes that the action of the drug on these muscles is analogous to its action on skeletal muscles in cases of myasthenia gravis, in which it has been used so effectively, the results here reported would tend to invalidate this analogy.

SUMMARY AND CONCLUSIONS

Prostigmine, a parasympatheticomimetic drug, has recently been used extensively in the treatment of tinnitus aurium.

Observations on 59 patients with deafness and tinnitus aurium made before, during and after a course of treatment with prostigmine are presented. All statements in reference to hearing are based on quantitative measurements carefully graphed by calibrated audiometers. Tests with the audiometer and tuning forks were repeated twice weekly.

The dosage was as follows: Prostigmine methylsulfate was administered hypodermically in a 1:2,000 sterile solution, the doses being 1 cc. (0.5 mg.). Twenty-five patients received injections daily; 10, three times weekly; 14, every other day, oral therapy with prostigmine bromide tablets being given as a supplement, and 10 patients received injections three times weekly, as well as ammonium chloride (enteric) capsules. Each patient received a minimum of thirty-five injections, except for 3 patients for whom the therapy had to be abandoned because of untoward effects.

The following results were noted: Of the 59 patients in the series, 26 (44 per cent) failed completely to show any change with the therapy, either subjectively or objectively; 4 (6.7 per cent) said that some improvement had occurred, but there was no audiometric evidence to substantiate the statement, and these patients attested the same sort of subjective improvement when physiologic solution of sodium chloride

was substituted for the prostigmine; 18 (30.5 per cent) stated that there was a similar subjective improvement, which likewise could not be substantiated with audiometric evidence and which proved transitory despite continuance of therapy, and 8 (13.5 per cent) had audiometric evidence of an 8 to 10 decibel improvement, which was transitory. Three patients (5 per cent) had a severe anginal syndrome, which finally responded to atropine therapy but which necessitated the abandonment of the prostigmine therapy.

The drug had absolutely no beneficial effect on the tinnitus and deafness of the patients with otosclerosis.

We regret to state that prostigmine proved of little, if any, value in the treatment of these patients with deafness and tinnitus aurium.

1915 Spruce Street.

SURGICAL CORRECTION OF COLLAPSED ALAR CARTILAGES

REPORT OF A CASE

CHARLES E. GURNEY, M.D.
PORTLAND, ORE.

This rather uncommon but definite clinical entity, known as collapse of the alae or, by the older writers, aspiration of the alae nasi (Laurens [1]) gives rise to partial or complete obstruction to nasal respiration. Although the symptoms may be extremely annoying to the patient, they can be entirely relieved by appropriate surgical treatment, the technic of which is described in the accompanying report.

This condition is characterized by extreme flaccidity of the lateral walls of the nose. Normally the musculocartilaginous structure of the alae, especially the lateral crura of the alar cartilages, is sufficiently rigid to keep the nostrils open, even under the stress of forced respiration such as accompanies exertion. When the walls of the nose lose their normal firmness, they have a tendency to move inward and outward with each phase of respiration. Barsky [2] refers to them as "flutter valves." During quiet respiration this may not give rise to symptoms, but under conditions of exertion such as heavy work, the nasal walls move medially far enough with each inspiration to come into intimate contact with the nasal septum. This results in partial or complete obstruction to the ingress of air, and the patient is forced to breathe through his mouth.

English literature throws little light on the cause of the condition, but paralysis of the nasal muscles, absence or weakness of the lateral crura (Sheehan [3]) and reversal of their normal curvatures (Foman [4]) have been mentioned as possible causes. Whatever the cause, the treatment is directed toward reenforcement of the nasal walls to prevent their collapse on forced respiration. Morrison [5] suggested either the

1. Laurens, G.: Oto-Rhino-Laryngology, translated by H. C. Fox, New York, William Wood & Company, 1922, p. 138.
2. Barsky, A. J.: Plastic Surgery, Philadelphia, W. B. Saunders Company, 1938, p. 149.
3. Sheehan, J. E.: Plastic Surgery of the Nose, New York, Paul B. Hoeber, Inc., 1936, p. 130.
4. Foman, S.: The Surgery of Injury and Plastic Repair, Baltimore, Williams & Wilkins Company, 1939, p. 733.
5. Morrison, W. W.: Diseases of the Nose, Throat and Ear, Philadelphia, W. B. Saunders Company, 1938, p. 60.

wearing of a suitable wire splint within the nostril or an operation which consists of cutting a small strip of cartilage from the nasal septum and implanting it in a subcutaneous pocket in the nasal vestibule. Sheehan [5] advised a rearrangement of the cartilages within the lateral nasal wall. Safian [6] recommended the use of a cartilage transplant and stated that if the perichondrium is left attached to one side of the cartilage, the latter will curl sufficiently to produce the desired contour of the nostril. Barsky described a method of transplanting aural or costal cartilage into the lateral nasal walls or into the septum. In the case reported here a costal cartilage transplant was employed, although the technic used was somewhat different from that usually described.

REPORT OF CASE

H. D., a man aged 54, a logger, complained of difficulty in getting sufficient air through his nostrils, especially when he did heavy work. The left nostril

Fig. 1.—Preoperative photographs: *A*, narrow pointed nasal tip and flat nasal walls; *B*, depression on the side of the nose at the level of greatest excursion during respiration.

offered greater resistance to breathing than did the right. His symptoms had been present for the past twenty-nine years, despite an operation on the nasal septum nineteen years ago, as it afforded him no relief. Although there had been little change in the severity of his symptoms during the past few years, the continual annoyance of having to breathe through his mouth while working forced him to seek relief at this time.

Examination.—The general physical examination showed nothing of importance. The nose appeared thin and somewhat pointed. The alar walls were flaccid, and their external surfaces were concave instead of convex, as they are normally (fig. 1 *A*). The lateral crura of the alar cartilages were present, but they had lost their firmness and did not hold the lateral nasal walls outward in the normal position. On quiet respiration the sides of the lose moved only slightly, but on

6. Safian, J.: Corrective Rhinoplastic Surgery, New York, Paul B. Hoeber, Inc., 1935, p. 98.

forced inspiration they were sucked tightly against the septum, causing complete obstruction in the nasal airways. The level of obstruction was most marked at a point approximately 8 mm. above the alar margin. On the outside of the nose

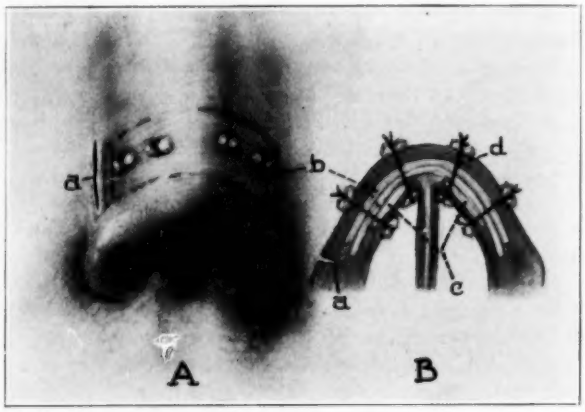

Fig. 2.—*A*, position of cartilage implant relative to the nose as a whole. *B*, relation of strips of cartilage to each other and also to neighboring structures: *a*, dermal incision through which the cartilage was inserted; *b*, cartilage implant; *c*, alar cartilage, and *d*, silk retention sutures tied over cotton pledgets.

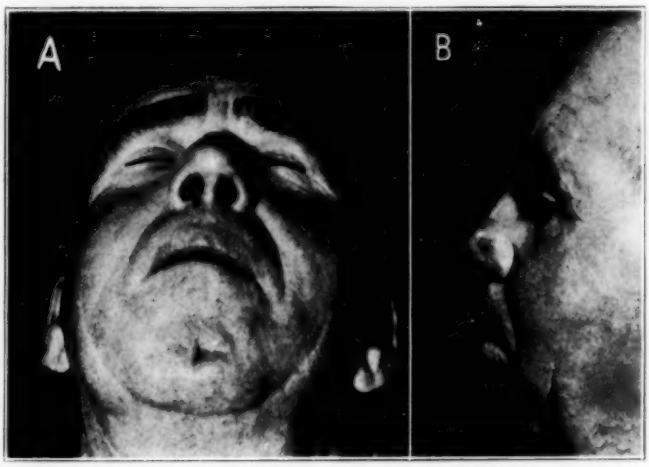

Fig. 3.—Postoperative photographs: *A*, rounded nasal tip and enlarged airway; *B*, convexity of lateral nasal wall.

and corresponding to this point was a definite depression in the skin, which represented the thinnest part of the alar wall (fig. 1 *B*). Although the septum was perforated (Wassermann reaction negative), it was straight and offered no unusual obstruction.

Operation.—With the region under local anesthesia, a segment of the seventh costal cartilage was removed from the right side. Two pieces of cartilage were then cut from this block, each piece being approximately 2 by 8 by 55 mm. The perichondrium was left attached to one side of each piece. Likewise with the region under local anesthesia, the skin over the entire nose was then undermined through a marginal incision just within each nostril. To facilitate the insertion of the cartilage, a small incision about 12 mm. in length was made through the skin on the right side of the nose close to and parallel with the cheek. One strip of cartilage was then inserted through the dermal incision, and by careful manipulation its forward end was carried over the bridge of the nose to the opposite side. The second piece was placed directly on top of the first, their flat surfaces being in contact. They extended laterally on each side to approximately the level of the distal end of the lateral crus. Their lower borders were about 5 mm. above the alar margin (fig. 2 *A*).

With the nostrils then held outward in their normal convex position, the relation between the cartilage strips, as well as that between them and the surrounding soft tissue, was maintained by two through and through sutures on each side of the nose (fig. 2 *B*). The incisions were closed with fine black silk, and a rather large soft rubber tube was inserted into each nostril. An adequate pressure dressing was applied over the bridge of the nose by means of a metal splint.

Course.—Except for a slight drainage from the dermal incision for the next week or two, the convalescence was uneventful and the final result entirely satisfactory. The patient was observed over a period of six months after the operation, and during this time there was no tendency for the alae to collapse even on forced inspiration. The nasal airways were adequate (fig. 3).

COMMENT

The question might justly be raised as to why two strips of cartilage were used instead of a single piece. It can be readily demonstrated that when two flat objects (pieces of thin cardboard for example) are held in contact with each other and then bent into a semicircle, a shift or sliding motion occurs between their opposing surfaces. If these surfaces are then securely fastened to each other while the strips are held in the semicircle, they will have a tendency to remain in this curved position. It is on this principle that the success of the described technic depends. Therefore it is essential that the through and through retention sutures and the pressure dressing remain in place sufficiently long to permit firm union to be established between the two strips of cartilage. In the case here reported the sutures were removed in five days, but an additional period of several days would do no harm. The rubber tubes in the nostrils and the pressure dressing over the nose should not be disturbed for a week except for observation.

Leaving the perichondrium attached to one side of the cartilage renders the latter less likely to be broken while it is being manipulated into position. A successful "take" of the cartilage is not dependent on the presence of the perichondrium.

A word of caution should be voiced regarding the adoption of this procedure for every patient with collapse of the alae. It will be noted that the rather slender and pointed nasal tip was converted into one which was wider and more rounded. In the case reported, the patient had no objection to this change. He was well satisfied to be able to breathe freely. But if a patient should possess a highly developed sense of pulchritude, he might seriously object to this rather notable change in the contour of the nasal tip. Since the psychologic makeup of each patient should be carefully studied before any plastic procedure is undertaken, the proper selection of patients should not be difficult.

EVALUATION OF VERTIGO FOLLOWING HEAD INJURIES

S. BAUMOEL, M.D.
AND
M. I. MARKS, M.D.
CLEVELAND

Dizziness is frequently encountered after head injuries. When associated with other neurologic signs, as it often is shortly after the trauma, it leaves no doubt concerning its organic origin. This discussion however will be limited to those cases in which months or even years after a head injury, frequently of a mild type, dizziness is the outstanding or the sole complaint. This dizziness almost always occurs either spontaneously or as a result of certain movements or positions.

Examination of the patients usually reveals no clinical signs to explain this complaint of dizziness, and therefore these patients are usually labeled as having some type of neurosis. The most common diagnoses are traumatic neurosis, traumatic hysteria, encephalopathy and traumatic encephalitis—obviously, all diagnoses of embarrassment. The physician's predicament is due to his omission of the examination of the only organ capable of producing this symptom, i. e., the vestibular apparatus. The examination of this sense organ, and this examination only, will ultimately decide the all-important question whether the dizziness is organic or psychogenic.

Dizziness is one of the most common complaints met with in the daily practice of medicine. It is, unfortunately, also one of the vaguest complaints, because the patient includes in it a multitude of sensations, most of which cannot be accepted as vestibular dizziness, such as a feeling of faintness or weakness, a darkening of the visual field and the peculiar discomfort produced by double vision.

With no attempt at elaborate and necessarily cumbersome definitions, it may be stated here that true vertigo is exclusively of vestibular origin and has definite characteristics. True, or vestibular, vertigo assumes two distinct forms: (1) turning dizziness and (2) tactile dizziness. Turning dizziness is the most common form found in disease of the

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From the Neurological and Ear, Nose and Throat Departments of Mount Sinai Hospital.

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vestibular apparatus and is the only form produced experimentally by turning or douching. It implies a rotation of the patient's surroundings or of his own body, and this rotation should be constant and in a definite direction. This type of dizziness is valuable as a focal symptom of diseases of the vestibular apparatus. Tactile dizziness includes erroneous impressions on the part of the patient as to the relative position of his body and the objects with which he is in actual contact. Brunner [1] thoroughly investigated tactile dizziness and distinguished two clinical manifestations: (1) erroneous impressions as to the position of objects within the tactile area—patients, complaining, for instance, in the attack of dizziness that the floor or the bed assumes a slanting position, and becoming frightened of falling out of bed; (2) erroneous impressions as to the position of the body, such as sensations of lateropulsion (Babinski and Nageotte [2]) and of laterotraction (Brunner [1]).

The description of dizziness as offered by the patients, however, is as a rule unsatisfactory and inconclusive, for the terrifying effect of an attack causes them to lose all ability of self observation. Yet even if the description were accurate, confirmatory evidence would still be necessary, since the symptom is purely subjective. Among the numerous symptoms of disturbance of the vestibular apparatus there is one which is as distinctly objective as dizziness is subjective, namely, nystagmus, an important and absolutely reliable clinical sign because it can never be simulated, is easily seen and permits of a more special localization within the extensive vestibular apparatus. Since vertigo and nystagmus are almost always associated in cases of vestibular disturbances, so that whenever there is a true vertigo nystagmus is sure to be found also, the simultaneous finding of nystagmus is confirmatory evidence that the dizziness is of vestibular origin.[3]

METHODS OF PROVOKING SPONTANEOUS NYSTAGMUS

Unfortunately the opportunity to witness an attack of vertigo is rare. We therefore provoke dizziness in order to observe the associated nystagmus.

The first step toward provocation is the so-called head movement test. In cases of lateral nystagmus we are at times able to bring out this nystagmus by moving the head rapidly in various directions. When this fails we attempt to produce a positional vertigo and nystagmus

1. Brunner, H., in Alexander, G., and Marburg, O.: Handbuch der Neurologie des Ohres, Berlin, Urban & Schwarzenberg, 1923, vol. 1.

2. Babinski, J., and Nageotte, J.: Rev. neurol. **10**:358, 1902.

3. The reverse of that dictum, i. e., that nystagmus is always associated with vertigo, is not always true. In cases of multiple sclerosis, for example, there is frequently nystagmus without dizziness.

by the following procedure:[4] First the patient is placed flat on his back with a pillow rolled under his neck. Then he is turned alternately on his right and his left side and finally on his back with the head overhanging. Frequently in the course of these procedures the patient excitedly exclaims that he is dizzy and that he is experiencing the sensation of which he originally complained. One can now invariably observe the incriminating nystagmus. During all examinations for nystagmus it is advisable, for accuracy as well as for convenience, to have the patient wear spectacles of plus 20 diopters, as recommended by Bartel. The purpose of these glasses is to eliminate fixation and by means of the magnification to facilitate observation of the finest nystagmic movements.

It is generally accepted that any second degree nystagmus,[5] even of the finest amplitude, not recognizable with the naked eye, is absolutely pathologic.

It should be particularly emphasized that the examination for positional nystagmus should never be omitted, for it is frequently the only objective finding in patients complaining of dizziness after more or less trivial head injuries, and by this examination a purely subjective symptom is objectified and the disability verified.

METHODS OF EXPERIMENTAL STIMULATION OF THE VESTIBULAR APPARATUS

Unfortunately, in a great many cases we are not successful in provoking positional vertigo and associated nystagmus, and then we must resort to experimental stimulation of the vestibular apparatus. Furthermore, experimental testing of the vestibular apparatus is essential in localizing an already established vestibular nystagmus.

This is not the place to go into the technical details of the various vestibular tests, but because of the confusion in the literature a few controversial points must be mentioned here. The chief confusion exists in regard to the method of employment and the interpretation of the caloric tests.[6]

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4. Seiferth, L. B.: München. med. Wchnschr. **83**:310, 1936.

5. The classification of nystagmus used here is Alexander's, based on the physiologic fact that nystagmus becomes intensified by a look in the direction of the quick component and that, on the other hand, it is decreased or inhibited by a look in the direction of the slow component. First degree nystagmus is visible only when the person looks to one side with the quick component to the same side. Second degree nystagmus is already noticeable when the patient looks straight ahead. Third degree nystagmus is one which can still be observed when the patient is looking to the side of the slow component.

6. Jones, I. H., and Fisher, L.: Equilibrium and Vertigo, Philadelphia, J. B. Lippincott Company, 1918.

It is frequently advocated that in carrying out the caloric test one should place the head straight forward or 30 degrees downward, with the horizontal canals in the horizontal plane. However, since the dynamic theory that the flow of endolymph produces the vestibular reaction is generally accepted, it is clear that such a position is the most unfavorable, at least for the horizontal semicircular canal, for any gradient in the endolymph flow is thereby abolished. It is true that the purpose of the caloric test is to stimulate one entire labyrinth, but it has been proved by several investigators, particularly Lorente de Nó,[7] that it is the horizontal canal which is mainly concerned in the production of caloric nystagmus. But if the horizontal canal is cut off from any possible stimulation by being placed absolutely horizontally, the effect of the caloric stimulation must be feeble, as it originates from the much less sensitive vertical canals. Furthermore, we have repeatedly demonstrated in our clinical work and on perfectly normal persons that when the horizontal canal is in this pessimum position the effect of caloric stimulation is feeble and frequently entirely absent.

We have further demonstrated that when the head is raised slowly from the horizontal position and approaches the optimum position of Brünings (60 degrees backward)[8] nystagmus of increasing intensity is seen and that when the head is lowered and brought back to the pessimum position the nystagmus becomes more and more feeble, often disappearing entirely when the horizontal canal again reaches the horizontal position, i. e., the position with the head 30 degrees forward.

The conclusion, therefore, is perfectly logical, that in performing the caloric test one should place the head in such a position as to produce the greatest gradient in the endolymph flow in the horizontal canal, i. e., 60 degrees backward.

In our experiments we employ the caloric tests, with hot and cold water, and the turning tests. The turning tests need not be discussed, as there is almost complete agreement about them as well as uniformity in their execution. In performing the caloric test we follow in the main the method advocated by Frenzel.[9] In short, having the head in the optimum position, we use water at 60 F. and 115 F. for five seconds, noting the latent period and the duration and character of the nystagmus.

RESULTS WITH EXPERIMENTAL STIMULI

The simultaneous presence of spontaneous nystagmus and vertigo clinches the diagnosis of a vestibular origin of the vertigo. When the

7. Lorente de Nó, R.: Tr. Am. Laryng., Rhin. & Otol. Soc. **42**:1936.
8. Brünings, W.: Ztschr. f. Ohrenh. **63**:20, 1911.
9. Frenzel, H.: Nervenarzt **4**:21, 1931.

attempt to obtain such nystagmus has failed, then experimental stimulation of the vestibular apparatus is the only remaining means of determining the real nature of this vertigo.

In general it may be assumed that any abnormal reactions obtained by experimental stimulation of the vestibular apparatus, whether of peripheral or of central origin, will demonstrate that the vertigo in question is a symptom of vestibular imbalance.

The peripheral disturbances causing dizziness following head injuries, such as fractures [10] through the petrous portion of the temporal bone, rarely fall into the scope of this discussion, for vertigo produced by traumatic destruction of a labyrinth lasts only a short time, about two or three weeks. But if the vertigo after such trauma does persist, as occasionally happens, the diagnostic difficulties as to its nature, organic or psychogenic, are greatly reduced by the history and nature of the injury and by the constant association with unilateral deafness, for if the labyrinth is affected by trauma there is always a simultaneous involvement of the cochlea, because of the greater vulnerability of that organ.

It must be borne in mind, however, that injuries severe enough to cause a fracture through the temporal bone may at times also cause concussion of the brain stem and thereby a central vertigo in addition to the peripheral. Such a seemingly complicated situation is readily clarified by the results of the experimental vestibular tests.

Generally speaking, qualitative changes in the responses to the experimental tests indicate central disturbances whereas quantitative changes are strongly suggestive of peripheral lesions. Some such qualitative changes are abnormal directions of the nystagmus, dissociation of the nystagmic movements in the two eyes as regards amplitude and frequency, inconstancy of the amplitude and frequency of the nystagmus in both eyes, a complete absence of the quick component with a deviation of the eyes in the direction of the slow component as well as others. However, the most important qualitative change and one frequently found after head injuries is the so-called tonus difference between the two vestibular systems.

Because of the importance of this phenomenon, the frequency with which it occurs particularly after head injuries and the rather rare allusion to it in the American literature, we should like to discuss it in greater detail.

Several authors, among them particularly Dusser de Barenne and de Kleyn,[11] in experimental work on animals, have made the observation

10. Grove, W. E.: Laryngoscope **49**:678 and 833, 1939.

11. Dusser de Barenne, J. G., and de Kleyn, A.: Arch. f. Ophth. **111**:374, 1923.

that after extirpation of one cerebral hemisphere the experimental nystagmus has been more pronounced toward the side on which operation was done, regardless of which labyrinth was stimulated and of what stimulus was used. Dusser de Barenne and de Kleyn called this phenomenon *Nystagmusbereitschaft,* or latent nystagmus, and drew the logical conclusion that it was independent of the peripheral labyrinth.

A few years later Vogel [12] found this phenomenon in his clinical work on man and gave it the name of "tonus difference."

After Vogel, other clinicians and investigators occupied themselves with this subject. They called the anomaly latent nystagmus because it behaves exactly like spontaneous nystagmus, for when the labyrinths of a person with spontaneous nystagmus are stimulated the experimental nystagmus is always more pronounced in the direction of the already existing spontaneous nystagmus because of the summation of the two nystagmi. In the other direction the experimental nystagmus would

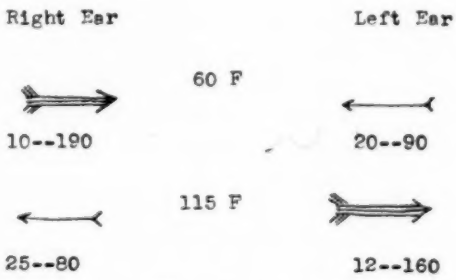

Fig. 1.—Characteristic tonus difference, i. e., difference between right and left nystagmus, revealed by experimental stimulation of the ears with cold (60 F.) and hot (150 F.) water. The number of lines and tails on the arrows in this and the following figures indicates the frequency and intensity of the nystagmus.

have to spend most of its energy in overcoming the spontaneous nystagmus before it could become manifest.

The characteristic features of a tonus difference can best be illustrated in the schematic record shown in figure 1. Douching of the right ear with cold water (60 F.) produces a nystagmus of great frequency and intensity to the left, beginning after a latent period of ten seconds from the start of irrigation and lasting one hundred and ninety seconds. Douching of the left ear with hot water (115 F.) produces a nystagmus in the same direction, of almost the same frequency, intensity and duration. On the other hand, douching of the left ear with cold water and of the right ear with hot water, produce much weaker responses, as seen in the diagram.

12. Vogel, K.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **23**:39, 1929.

This means that the nystagmus to the left is predominant, and it becomes clear at once that this predominance cannot be due to hyperirritability of one ear or the other but is due rather to a central tendency controlling the intensity of the nystagmus in one particular direction, for if one peripheral labyrinth were hyperirritable or hypoirritable it would, of necessity, be so to all stimuli. It is the central vestibular apparatus that distributes the various stimuli coming from each labyrinth to either side of the vestibulo-ocular reflex tracts, composed of the posterior longitudinal bundle and the substantia reticularis (Lorente de Nó[13]). Each of these tracts receiving stimuli from both Deiters nuclei determines the direction of the nystagmus to its own side. If there is a disturbance in these tracts the resulting imbalance expresses itself in the predominance of the nystagmus to one or the other side. This difference between right nystagmus and left nystagmus has been expressed in terms of tone, hence the name tonus difference.

It should be added here that we also employ the rotation test in every case, first because it is the more physiologic stimulus and secondly because we have found that in cases of tonus difference it conforms to the findings of the caloric test and thereby accentuates this abnormality. Some authors employ also galvanic stimulation[14] of the labyrinth for the purpose of detecting or confirming a tonus difference, but for routine work one may dispense with both the galvanic and the rotation test. We must emphasize, however, the necessity of performing the caloric test with both hot and cold water in order not to miss the all-important tonus difference.

This tonus difference is the abnormality found most frequently in our series of cases of vertigo after head trauma. A few examples are presented.

REPORT OF CASES

CASE 1.—J. D., a white man aged 38 years, when first seen by us in August 1939 gave the following history: On Feb. 1, 1932 he was struck by a blackjack over the right side of the head. He was unconscious for several hours and after recovering consciousness felt a sharp pain in the back of the head, was extremely dizzy and was bleeding from the right ear and the nose. He was taken to a hospital, where roentgen examination revealed a fracture involving the petrous portion of the right temporal bone. Because of some psychotic symptoms which developed at the hospital, he had to remain there for about five weeks. After leaving the hospital, he still complained of severe headaches and dizziness in attacks and was nervous and irritable. Because of these symptoms, an exploratory craniotomy was done in an unsuccessful attempt to find a suspected subdural hematoma.

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13. Lorente de Nó, R.: Die Labyrinthreflexe auf die Augenmuskeln nach einseitiger Labyrinthexstirpation nebst einer kurzen Angabe über den Nervenmechanismus der vestibularen Augenbewegungen, Berlin, Urban & Schwarzenberg, 1928.

14. Koch, J.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **137**:105, 1934.

In August 1939, when first seen by us, the patient's complaints were as follows: Without any apparent cause, for instance while walking in the street or even when at rest at home, he would suddenly feel dizzy. Objects would turn around him mostly to the left side, and on many occasions this turning became so severe that he had to hold to a fixed object to avoid falling. At times he actually fell, and on several such occasions injured himself, once fracturing several ribs. During these attacks he was always nauseated and sometimes would vomit profusely. The individual attacks lasted about six to eight hours, but for several days afterward he would feel "all washed out."

Furthermore, the patient complained that frequently he was awakened at night by severe palpitation of the heart together with definite air hunger; on such occasions, he said, he would get panicky and run out of the house. These attacks had been occurring irregularly, sometimes once every two or three weeks and then again two or three times during one night. He had been irritable and moody and at times depressed, at which times he would talk to no one. He stated further that it had been difficult for him to apply himself to any kind of work because of the inability to concentrate for any length of time.

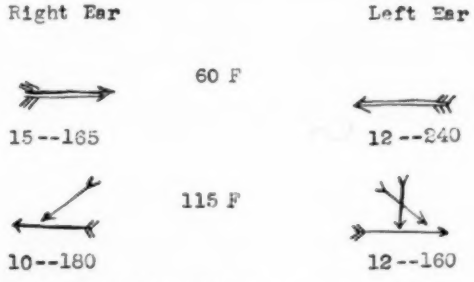

Fig. 2.—Tonus difference of the patient in case 1. When the ears were irrigated with hot water, there were frequent changes in the quality of the nystagmus. The small arrows denote variations in direction of the nystagmus. All these variations are characteristic of involvement of the central vestibular apparatus.

The past history and family history were irrelevant. The patient is married and has several children.

Examination.—The patient was a well developed and fairly well nourished white man, 38 years old. He was perfectly rational and well oriented but appeared somewhat dreamy and was slow in responding to questions. His memory as to the details of the accident and the subsequent illness was somewhat defective, and occasionally he made contradictory statements. Physical examination revealed the following positive signs: There was a small triangular depression in the right temporoparietal region of the skull. The reflexes in both lower extremities were somewhat exaggerated, and some suggestion of a bilateral Babinski sign was observed. There was past pointing with the right hand toward the right, as well as a positive Romberg sign with a tendency to fall toward the right. Marked positional nystagmus was noted when the patient turned the entire body from the supine position to either side.

The nystagmus to the right, when he turned onto the left side, was more pronounced and of the second degree. The latter position simultaneously evoked,

in addition to the second degree nystagmus, an attack of severe dizziness. The patient was terrified and suddenly cried out, "Doctor, this is just what I feel when I am dizzy."

Application of the various experimental tests to the patient's vestibular apparatus produced a definite tonus difference to the right, i. e., the nystagmus to the right was both qualitatively and quantitatively predominant over the nystagmus to the left. The reactions to stimulation by water are diagrammatically represented in figure 2.

The results of the turning tests were as follows: Turning to the right produced nystagmus to the left for thirty-five seconds, while turning to the left produced nystagmus to the right for forty-five seconds, the frequency and intensity in both instances being approximately that produced by douching with cold water (fig. 2).

The past pointing, the positional nystagmus, the positive Romberg sign and the tonus difference of the nystagmus elicited by both the caloric and the turning test point definitely to a disturbance of the vestibular apparatus in the central distribution within the brain stem. These strictly objective findings at once explain the attacks of dizziness and put them, beyond any doubt, on an organic basis.

These findings also objectify the various psychic and mental disturbances this patient presented. Because of the failure to examine the vestibular apparatus, the mental disturbances were interpreted by many as psychogenic.

Case 2.—H. P., a white man aged 39 years, when first seen by us in February 1939 presented the following history: On January 1935 he fell from a ladder and struck his head against a machine, sustaining a contused, lacerated wound on the right side of the head. He was unconscious for about one-half hour and was taken to a hospital, where several sutures were placed in the scalp wound. Immediately after the accident he complained of dizziness, headache and rushing of blood to the head. In spite of these complaints, however, he returned to work. The symptoms became so aggravated that he was unable to perform his work and was therefore discharged. About this time he was examined by several physicians, who stated that while it was likely he had suffered concussion of the the brain, the persisting symptoms of dizziness and headache were probably neurotic. About one and one-half years after the accident the patient obtained employment as a cherry picker; while on a ladder he suddenly became extremely dizzy and fell, sustaining fractures of the left arm and left leg.

When first seen by us in February 1939 the patient complained of dizziness on the slightest movement of the head. This dizziness assumed extreme intensity, he said, when he turned quickly in any direction, and on several occasions he had fallen to the ground. The dizziness was always accompanied by nausea, at times also by vomiting. Occasionally he would have an attack while walking on the street and would then have to stop and lean against some fixed object. Even turning during his sleep would precipitate a dizzy spell that would awaken him.

Examination.—The results of physical examination, including that of the nervous system, were entirely normal. The vestibular apparatus alone presented abnormal findings, which were as follows: Spontaneous nystagmus of the first degree to the left and at times also slight nystagmus of the second degree, which, however, could be detected only with the aid of Bartel's glasses. Figure 3 shows the reaction to caloric tests.

The patient in this case presented, as is strikingly shown in figure 3, a typical tonus difference. Only an examination of the vestibular apparatus could have revealed the true nature of this patient's complaint and thereby have spared him the unnecessary disabling injury.

CASE 3.—J. S., a white man aged 33 years, was seen by us on Aug. 5, 1936, and presented the following history: On May 8, 1936 he fell from a scaffold 40 feet (12.19 meters) high to a cement floor. Immediately after the fall he was unconscious for several hours and was taken to a hospital, where he remained for four weeks. When he regained consciousness, he felt extremely dizzy and

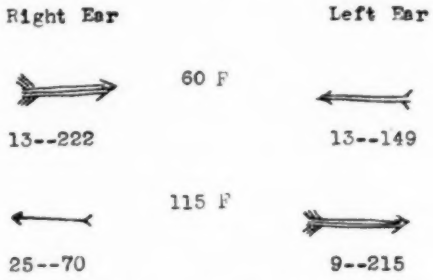

Fig. 3.—Tonus difference of the patient in case 2, as revealed in caloric tests.

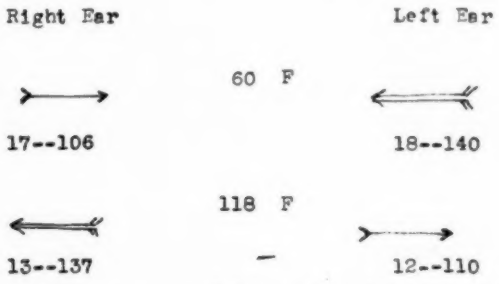

Fig. 4.—Tonus difference as revealed by early caloric tests (Aug. 5, 1936) of the patient in case 3.

vomited repeatedly some bloody material. During this dizzy spell he had the sensation that the entire room was turing around him, also that his bed was tilted and that he was in danger of falling out. After this first attack he felt only slightly dizzy, particularly when trying to raise his head. The next severe attack of vertigo occurred on getting out of bed in the morning after he had been home for two weeks, i. e., about six weeks after the injury. This attack lasted about one-half hour. He did not vomit, but had to sit down and hold to some fixed object to keep from falling. The next morning he had another attack just as severe, also lasting one-half hour, and at noon of the same day still another severe attack. He recalls definitely that this last attack occurred when he suddenly raised his head to look at his baby. After this he would have somewhat milder attacks daily for about two weeks; then they came on at greater intervals, but occasionally with the original severity.

Examination.—On Aug. 5, 1936 the results of physical examination, including that of the nervous system, were entirely normal with the exception of fairly marked deafness of the left middle ear. Examination of the vestibular apparatus showed a spontaneous first degree horizontal nystagmus to either side. The reactions to the caloric tests are shown in figure 4. These reactions showed a disturbance of the central vestibular apparatus in the form of a tonus difference.

On May 20, 1938 the patient presented himself for reexamination. At this time his complaints were negligible, for only on brusque movements would he occasionally experience mild vertigo. Figure 5 shows the results of caloric tests on this occasion.

This case has been included here because of an important feature, namely, a functional restitution of the vestibular apparatus, as evidenced by the results of the later caloric tests (fig. 5). In spite of his severe injury this patient's condition was also diagnosed as neurotic simply because of absence of any abnormal neurologic findings.

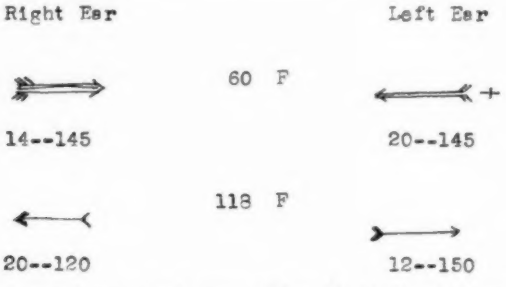

Fig. 5.—Reaction to later caloric tests (May 20, 1938) of the patient in case 3, illustrating restitution of the vestibular function.

COMMENT

It is difficult to understand why the examination of the vestibular apparatus is so frequently omitted in the face of dizziness as the presenting complaint. No physician would neglect to examine the eyes in a case of disturbance of vision or the ears in a case of impairment of hearing. Since the majority of the cases under our discussion involve some sort of liability compensation, great damage is being done to these patients by labeling them neurotic when a proper examination would establish a perfectly just claim. The vestibular tests have been greatly neglected because they have been somewhat disappointing as a source of diagnostic information. One frequently hears also the complaint that these tests are too time consuming, out of proportion to the possible diagnostic benefits derived from them. We believe we have conclusively shown that for the condition under discussion these tests are the only available means of arriving at a diagnosis, and we do not hesitate to place the importance of their application in cases of this condition next to the almost pathognomonic significance of vestibular tests in cases of acoustic tumor.

The abnormal findings alluded to in our discussion do not necessarily denote structural changes within the vestibular apparatus; on the contrary, in the majority of our cases the changes were functional. In contradistinction to "psychogenic," we apply the term "functional" in the same sense as we do with regard to conditions like migraine or epilepsy. Since both the complaint of dizziness and the abnormal vestibular findings are in most instances regressive, usually disappearing months or a few years after the trauma, we are led to believe that the cause of these disturbances is circulatory, similar to what we assume to exist in cases of concussion of the brain. Evidence has been accumulating in recent years to show that the brain stem is frequently involved with the rest of the brain in cases of head injuries producing the clinical picture of concussion.

SUMMARY

Patients complaining of vertigo some time after head injuries are discussed. The characteristics of true, or vestibular, vertigo are dealt with.

The vestibular nature of vertigo is being recognized by observations of the nystagmus which is almost always associated with it.

The associated nystagmus may be spontaneous and then observed during an attack of dizziness, or both nystagmus and dizziness may be provoked by such stimuli as head movements and various body positions.

If we are not successful with those methods, we proceed to the various experimental tests.

Some significant errors in technic of the vestibular tests are mentioned.

Among the results of the vestibular tests tonus difference is emphasized as of special diagnostic significance.

A plea is made for the importance of the application of the vestibular tests in cases of vertigo after head injury when otherwise normal findings appear.

THROMBOSIS OF THE CAVERNOUS SINUS

ERRETT PACE, M.D.
PADUCAH, KY.

John Abercrombie discovered the first thrombosis of a cavernous sinus to be reported. He made the finding during an autopsy, and it was reported by Duncan [1] in 1821. Clinical reports were also made in 1839, by Vigla (cited by MacGuire [2]). MacGuire [2] stated that in 1923 not many fatal cases had been reviewed in the literature and that the seriousness of the condition was not generally appreciated at that time. Before 1923 only two contributions of consequence could be found, one of which was by Bullock.[3] Bullock ligated and divided both facial veins before treating a serious carbuncle involving the upper lip, from which the infection had spread, causing the eyelids to become red and swollen shut. This appears to be the first case in this country in which the patient was saved by ligation of the facial veins.

ETIOLOGY

Among the many causes of thrombosis of a cavernous sinus are marasmus, trauma and infection. The marasmus type usually occurs in the very old or the very young who are afflicted with debilitating conditions. Involvement of the cavernous sinus is practically never primary but follows that of the longitudinal or the lateral sinus.

The most common form is septic thrombosis secondary to thrombophlebitis of some of the afferent or efferent veins, but it may be primary in the cavernous sinus, owing to direct extension from basilar meningitis, abscess of the postorbital space, suppurative sphenoiditis or suppuration of the petrous portion of the temporal bone.

From the Pediatric Service, Riverside Hospital.

Thesis submitted to the Faculty of the Graduate School of Medicine of the University of Pennsylvania in partial fulfilment of the requirements for the degree of Master of Medical Science (M. Sc. [Med.]) in regard to graduate work in otolaryngology.

1. Duncan, A., Jr.: Contributions to Morbid Anatomy, Edinburgh M. & S. J., 1821, vol. 17.

2. MacGuire, D. P.: Internat. J. Med. & Surg. **48**:114-116, 1935.

3. Bullock, W. O.: Surg., Gynec. & Obst. **14**:156-158, 1912. Sebileau, P.: Bull. et mém. Soc. de chir. de Paris **38**:123, 1912.

FREQUENCY

Although not a surgical oddity, thrombosis of a cavernous sinus is a rare condition.

Chisolm and Watkins [4] stated "Thrombosis of the caverous sinus is a very rare condition. Only 8 cases were found by us in 50,000 of the surgical records of the Johns Hopkins Hospital."

Irish [5] made a survey of 34 cases, in which, he stated, the left cavernous sinus alone was thrombosed in 5 instances and the right in 6; both cavernous sinuses were thrombosed in 10, and involved in other thrombotic processes (including the ophthalmic veins in 2 instances in 6 other cases. In 12 instances miscellaneous and multiple sinuses were thrombosed, the involvement including the superior veins, the transverse, sigmoid, basal veins and the middle cervical veins.

PATHWAYS OF INFECTION

1. Bailey [6] stated that infection occurs by way of the facial veins. Eagleton [7] said that infections around the lip, eyelid, nasal orifice, teeth or anterior part of the mouth travel by retrograde thrombophlebitis through the angular or facial venous plexus, into the orbit and then through the ophthalmic vein to the cavernous sinus.

2. Infections around the posterior teeth, the jaw or the maxillary antrum and less frequently those in the pharynx or tonsil extend through the pterygoid fossa with its numerous anastomoses from the pharyngeal plexus, through the foramen ovale and the deep facial and inferior ophthalmic veins. When the infection causing thrombosis of a cavernous sinus originates in the tonsil, it usually travels through the internal jugular vein and then the inferior petrosal vein; however, there are records of direct extension from the tonsil into the pterygoid plexus, in which the tonsillar veins anastomose.

3. The aural type enters through the inferior petrosal vein or from deep cells at the apex of the petrous pyramid.

4. The sphenoid, or mesial, infection reaches the mesial portion of the sinus by caries of the sphenoid bone or thrombosis of the veins which ascend through the roof of the sphenoid sinus.

5. Thrombosis of the carotid venous plexus may extend into the cavernous sinus by a microscopic pathway. Here a jugular route from

4. Chisolm, J. J., and Watkins, S. S.: Twelve Cases of Thrombosis of the Cavernous Sinus, Arch. Surg. **1**:483-512 (Nov.) 1920.

5. Irish, C. W.: Ann. Otol., Rhin. & Laryng. **47**:411-436, 1938.

6. Bailey, H.: Lancet **1**:1053, 1931.

7. Eagleton, W. P.: Cavernous Sinus Thrombophlebitis, New York, The Macmillan Company, 1926.

either the ear or the throat extends through the internal carotid canal into the skull, and thus reaches the anterior portion of the cavernous sinus.

6. Although anatomically and clinically distinct, all six types may pass from one to another because of the free anastomotic communication of the venous radicals.

ANATOMIC RELATIONS

Batson,[8] in his discussion of a case reported by Barnshaw, stated:

About the upper lip, the nose and the orbit, the venous blood may travel to the neck either superficially through the superior ophthalmic vein, or through the cavernous sinus and finally to the jugular bulb. Since here there are no valves, the flow in the veins is determined by the propulsion from the capillary side, by changes in posture, and by impediments to flow, such as those produced by the pressure of an oedema. Therefore, an oedema may cause an infected blood stream to pass through the cavernous sinus, with the subsequent involvement of the meninges. When the infection is in the lower part of the face, oedema may prevent the course of blood through the superficial channels, but an alternate deeper pathway can carry the stream. This pathology is through the pterygoid plexus of veins, and it lies outside of the cranial cavity. To my mind this anatomical difference in the relationship of the alternate pathways, the one through the meninges, the other outside the skull, determines the severity of the infections in the upper and lower facial areas.

Faulkner[9] wrote as follows:

The anatomical connections of the cavernous sinus are well known, and from such knowledge we can postulate the areas of local infection from which a thrombus may extend to the sinus. They may be classified according to the venous area of origin, though these areas overlap to some extent. Thus we have:

1. Anterior foci with infection spreading through the angular, supraorbital and supratrochlear veins to the ophthalmic veins.

2. Internal foci through the ethmoidal veins or spreading through the walls of the sphenoid sinus.

3. Inferior foci with spread of infection through the pterygoid plexus or by direct proximal infection of the internal jugular through lateral sinus and petrosals.

4. Posteriorly by extension from lateral sinus through the petrosals. . . .

The anterior group of focal infections comprises such lesions as boils and carbuncles about the lip or ala nasi, and infections of the eye-lids. . . .

The internal group includes sinus infections (among which are those following intranasal operation).

The inferior group embraces: peritonsillar abscess; infection of operative field after tonsil operation; infections of superior maxilla, usually after extraction of teeth or primary osteomyelitis. Deep cervical abscess may infect the sinus through the pterygoid plexus or by infection of the jugular vein, thence through the lateral sinus and petrosals.

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8. Batson, O. V., in discussion on Barnshaw.[15]
9. Faulkner, E. R.: Surg., Gynec. & Obst. **52**:474-478, 1931.

The incidence of involvement from these foci of infection, he wrote (citing Dorland Smith), is approximately as follows: "ear, 40 per cent; orbit and face, 35 per cent; mouth and throat, 13 per cent; nose, 9 per cent; other causes, 3 per cent."

Bowers [10] made a somewhat contradictory statement that as a complication of mastoiditis, thrombosis of a cavernous sinus is a rare condition. This is clearly evidenced on a careful search of cases in which sinus thrombosis was secondary to mastoiditis.

ANATOMY

No thorough understanding of any condition can be obtained without a knowledge of the anatomy. This is especially true in the case of disease of the cavernous sinus, which has anatomic peculiarities found in no other organ unless possibly the corpus cavernosum of the penis, from which it received its name.

The cavernous sinuses are about 3 cm. in length and 1 cm. in diameter and are located one on each side of the sella turcica. They are contained within the reduplications of the dura mater that form the lateral walls of the osseomembranous pituitary fossa, and are directed cephalocaudally, extending from the sphenoid fissure in front of the apex of the petrosa to the rear, where they terminate by dividing into the superior and the inferior petrosal sinus. They lie close to the roof of the sphenoid cavity and are separated from it by a thin bony partition.

The internal carotid artery, the abducens nerve, a sympathetic plexus and numerous trabeculae all tend to reduce the lumen of the cavernous sinus.

The oculomotor nerve, the trochlear nerve and the ophthalmic and maxillary divisions of the trigeminal nerve are found in the outer wall. The ophthalmic division enters the lower portion of the outer wall as the sinus passes over the mesial portion of the gasserian ganglion. The trochlear nerve lies just above the ophthalmic and follows a slightly oblique course through this wall of the sinus; on arriving at the sphenoid fissure it is slightly dorsal to the oculomotor nerve. The oculomotor nerve enters the outer wall of the sinus in a manner similar to that of the trochlear nerve, but more dorsally and passes close by and ventral to the posterior clinoid process. Its direction is slightly downward, so that in its course forward it passes slightly mesial to the trochlear nerve and appears slightly below this nerve at the sphenoid fissure. The ophthalmic and the oculomotor nerve divide into their branches in the anterior one fifth of the sinus.

The outstanding feature of the cavernous sinus is undoubtedly the network of trabeculae that transverse it. These are formed of fibrous

10. Bowers, W. C.: Laryngoscope **27**:372-376, 1927.

tissue and vary in size; the smallest are almost invisible, while the largest are as thick as a small nerve. The trabeculae run horizontally, longitudinally and diagonally—in fact, in almost every direction without any set order. They are attached to the internal carotid artery as well as to the abducens nerve as these pass through the sinus. All the bands are covered with endothelium. Connecting circulation is found in the superior and inferior opthalmic veins, which bring blood from the forehead, the supraorbital region of the nose, the eye and other sections of the upper part of the face. The inferior and anterior cerebral and middle meningeal veins bring blood from the cerebrum and meninges. The sphenoparietal sinus brings blood from the dura mater. The orbital veins unite at the root of the nose to form the angular vein, which in turn becomes the facial vein. The anterior and posterior ethmoidal veins, the ciliary veins from the tissues of the orbit and a tributary from the frontal diploe all enter the superior ophthalmic vein. An important branch of the inferior ophthalmic vein communicates with the pterygoid plexus through the sphenomaxillary fissure.

Blood is carried from the sinus through the superior and the inferior petrosal vein into the lateral sinus.

A plexus of veins around the carotid artery carry blood to the jugular bulb.

Other small veins pass from the under surface through the foramina rotundum, ovale and lacerum medium and the canal of Vesalius to the pterygoid plexus.

PHYSIOLOGY

The veins that drain the surrounding tissues into the cavernous sinus are distinguished by a special anatomic peculiarity: they are destitute of valves; at least the valves are so rudimentary that they fail to prevent the free passage of blood in either direction. This peculiarity is also found in all the intracranial veins as well as in those of the pterygoid plexus and in the small veins joining the latter with the cavernous sinus. It has been shown that the direction of flow in the intracranial veins and in the plexuses just mentioned is frequently changed, owing to gravity, muscular exertion and the respiratory cycle.

Deep inspiration tends to draw blood from the jugular vein into the large veins of the chest, which in turn draws blood from the brain by way of the large venous sinuses. However, this is compensated for to a great extent by the oblique way in which the tributaries enter into the lateral sinus, by the division of blood into two trunks at the torcular Herophili at right angles to the original flow and by the tortuous course through the sigmoid sinus with considerable narrowing at the jugular foramen before it enters the jugular vein.

The only other exit of blood from the brain is through the cavernous sinus. This carries the blood the same as other venous sinuses, but

anatomically it is so different from any other venous channel that much speculation has been made on its specific function.

The enormous number of criss-cross trabeculae in the sinus apparently occlude the lumen and tend to make it worthless as a conveyor of blood. In places the bands are so numerous as almost to filter out the corpuscles. Undoubtedly, this arrangement by nature is not without purpose. Probably the most reasonable explanation of the anatomic peculiarity is that it controls blood pressure within the brain and prevents too rapid depletion of blood in that organ during the respiratory phase. This same principle could be applied to the effects of muscular strain. Sudden violent contraction of muscles in an area tends to force the blood away and temporarily to increase the volume at distant points.

The proper nourishment of the brain depends on slow circulation of the blood. Also the blood flow in the carotid plexus must be slow to allow for the osmosis into the ventricles by which the spinal fluid originates. A rapid flow of blood through the large venous sinuses would depend on a high degree of pressure, which would prevent the pacchionian bodies located in and around the sinuses from passing the cerebral fluid back into the venous sinuses, thus completing the cerebrospinal circulation.

Therefore it may be reasonable to assume that the cavernous sinus has been peculiarly designed by nature to keep the hydrostatic pressure in the brain normal at all times.

PATHOLOGY

The anatomic construction of the sinuses admirably predisposes them to thrombosis. They are rigid and relatively wide, with consequently slow circulation; in the cavernous sinus there is the interesting fibrous network of its lumen, already considered. It is difficult to explain why the cavernous sinus is not more frequently thrombosed than the lateral sinus, unless the pathologic condition adjacent to the latter is in some way different. The sphenoidal air cell is near the cavernous sinus, but unlike what is observed of the mastoid and the lateral sinus, an infection of it does not extend to the adjacent sinus except in rare cases. Probably this is due to a better natural drainage from the sphenoid than from the mastoid cells.

Thrombosis of the cavernous sinus may be present without any symptoms. If the vein is not involved, a good collateral circulation would prevent any edema, ecchymosis or proptosis of the eye, since the venous blood from the eye can flow from the ophthalmic vein reversely into the angular and the facial vein. The thrombosis is merely a protective reaction. There should be no paralysis of the third, fourth or sixth cranial nerve, for there would not be enough pressure to paralyze them.

Thrombophlebitis gives marked and rapidly advancing symptoms. This spreads to adjoining veins, which is nature's way of preventing the infection from entering the circulation. Increased inflammation and pressure paralyze the third, fourth and sixth cranial nerves and give the typical ophthalmic findings of paralysis and loss of motion. As soon as the phlebitis reaches the inferior cerebral veins which run into the cavernous sinus, meningitis develops. These veins are without fibrous sheaths, and as they run through the chiasmal cisterna the infection spreads through the basal cisterna and over the cortex.

Infection in the sinus comes from foci situated on the face, lips, eyelids, mouth and throat, from which the venous drainage either directly or indirectly passes through the sinus. These channels are in many cases involved in thrombophlebitis, but it takes time for even thrombophlebitis to extend from the angular to the facial vein, then to the jugular vein, through the sigmoid sinus and then by way of the inferior petrosal vein to the cavernous sinus. In my opinion, however, this does not explain some of the rapidly developing infections. In some cases paralysis of the third, fourth and sixth nerves is produced within twenty-four to thirty-six hours. Then, is it not possible that an infected embolus may lodge in the network of the lumen and set up an infection much more quickly than is possible with thrombophlebitis?

When this clot breaks down and the virulent infection is thrown into the circulation, high fever and chills develop. The lack of valves in the veins of this area is considered contributory to the spread of the infected material to surrounding tissues. These veins are large, they do not collapse easily and the blood flow is slow—all of which contributes to frequent lodging of septic emboli. It is thought by some authorities that under certain conditions, such as deep respiration or muscular exertion, there may be a reversal of the normal direction of blood flow. Any squeezing, incision or trauma to an infected area may be the means by which a piece of infected clot is broken off and set free in the blood stream, to be caught in the cavernous sinus.

SYMPTOMS AND DIAGNOSIS

Benedict [11] stated:

> In thrombosis of the cavernous sinus the ocular symptoms are dominant, and in 10 per cent of cases of lateral sinus infection ocular symptoms are found. With the exception of these conditions, thrombosis and its sequelae lie outside of the field of ophthalmic practice. Nevertheless the differential diagnosis of sinus thrombosis is complicated and ocular signs and symptoms are of considerable aid if properly interpreted. Exophthalmus, oedema of the orbit and lids, progressive external phenomena and dimming of the optic nerve are the most frequently mentioned pathological conditions of the eye that are to be looked for in a case of suspected thrombosis.

11. Benedict, W. L.: Surg., Gynec. & Obst. **52**:464-470, 1931.

The significance of these changes may be decisive if there are other grounds for this conclusion.

Complete thrombosis of a lateral sinus thus mechanically obstructing the flow of blood may not produce characteristic changes about the eye. Resection of the jugular vein is done without noticeable change in the circulation of the orbit. This is not true of thrombosis of the cavernous sinus, which results in passive congestion with exophthalmos, swelling of the lids, impairment of motion of the eye and retinal venous congestion with edema of the head of the nerves. Other conditions may present these symptoms in varying degrees—for instance, periostitis, orbital cellulitis, parophthalmitis, tenonitis, tumors, diseases of the nasal accessory sinuses and chronic inflammatory conditions. A history of mastoiditis or of acute infection of the nose or paranasal sinuses would probably indicate thrombosis of a venous sinus, while without this infection the cause of exophthalmos would be looked for in the orbit.

Proptosis and swelling of the lids associated with septicemia in cases of infection about the head should cause one to suspect thrombosis of a cavernous sinus.

Jansen proved by autopsy that purulent thrombosis of the cavernous sinus is possible without the presence of any change in the fundus or any impairment of visual acuteness or of the function of the ocular muscles.

The blood count is usually high (20,000 to 40,000), and the spinal fluid is usually cloudy with a high cell count. Blood cultures are positive in the majority of cases. Usually both eyes are involved, the second within forty-eight hours after the first. Retinal edema, dilatation and tortuosity of vessels of the retina, retinal hemorrhages and sometimes slight choking of the disk, clouding of the media and corneal opacity usually occur within a few hours. Edema of the upper lid may completely close the eye. When the pterygoid plexus is thrombosed, there may be edema of the pharynx and tonsils on the same side. Edema of the skin over the mastoid process and of the neck may appear when the lateral and the jugular vein are involved, and of the face when the facial vein is involved.

Dilatation of the pupil is due either to stimulation of the sympathetic nerve or to paralysis of the oculomotor nerve. Ptosis is usually caused by paralysis of the oculomotor nerve but may result from edema of the upper lid. Fixation of the eye takes place early in the disease, owing to paralysis of the oculomotor, trochlear and abducens nerves as a result of pressure on them as they pass through the sinus. Pain in and above the orbit are frequent early symptoms. Meningitis produces a more severe and general pain. Trismus, which is probably due to irritation of the mandibular division of the trigeminal nerve, is rare but has been

reported. Although there is usually great loss of vision, it may be retained even when the condition is severe.

General symptoms are a septic temperature, a rapid, small and thready pulse, chills and sweats. There is fever temperature (100 to 106 F.) with marked depressions. Vomiting, delirium and coma may occur. Meningitis frequently develops. Pulmonary embolism, infection of the spleen, kidney or liver as well as septic embolism in various parts of the body must be differentiated from erysipelas. Eagleton [7] described acute and chronic types. The acute type gives the usual symptoms of obstruction to a large sinus, while, owing to the gradual compensation through collateral circulation, the chronic type produces no signs of venous obstruction.

Paralysis of the third, fourth and sixth nerves is due to pressure on them as they pass through the sinuses.

Paralysis usually appears late in the cellulitis except in the fulminating type, accompanied by rapid swelling and proptosis of the lid. Acute inflammatory conditions of the orbit, such as follow acute diseases of the nasal sinuses, are seldom bilateral—a valuable point of distinction. Bilateral edema of the lids is common when infection is limited to one orbit, but paralysis and proptosis involve only one eye. The other becomes involved within one to five days. When both orbits become involved, there is little doubt but that the cavernous sinus is infected unless the swelling persists while the general condition of the patient improves, for it is believed that comparatively few patients who have thrombosis of a cavernous sinus get well; inflammatory diseases of the orbit, on the other hand, are seldom fatal. In the early stages it is often impossible to get sufficient cooperation from the patient to enable one to make a certain diagnosis.

Thrombosis of the cavernous sinus is usually first suspected after the appearance of swelling of the lids and exophthalmos. If the retinal veins are distended and the disks edematous, the evidence is more conclusive.

PREVENTION

Prevention of thrombosis of a cavernous sinus is of necessity limited to the period before the bacteria have entered the blood stream. Patients should be instructed that any trauma or manipulation around the face, e. g. about the mouth, is dangerous. Pulling hairs from nostrils and squeezing pimples on the face are among the most frequent etiologic factors in opening channels for infection to enter the venous system. Many vessels have been ligated for prevention, but careful anatomic study will demonstrate that there are many routes by which the bacteria may travel, thus making ligation futile.

Bailey,[6] advocated ligation of the angular vein.

Batson,[8] in a discussion of a case reported by Barnshaw, made the following statement:

> The multiple channels in the venous system preclude the possibility of a single ligation arresting the progress of a diseased blood stream flow. In my experimental (post mortem) injections, it has been impossible to prevent the filling of the cavernous sinus even after extensive ligations on the face have been performed. This would make it seem that any virtue that the ligation is supposed to possess may come from the incision which acts as an avenue for drainage.

Practically every vein of the face and neck has been ligated for the prevention of spread to a cavernous sinus, but in no case has ligation proved worth while, owing to free anastomosis. Most incisions and manipulations prove harmful, serving only to open channels of infection and loosen septic clots that spread by way of the blood stream.

TYPES OF OPERATIONS

Operative procedures in the treatment of this condition are the products of some of the most brilliant minds and are clever from an anatomic standpoint. These operations have undoubtedly proved successful in some instances, but in general they have left the problem unsolved.

The operative procedures are of four types: (1) the Hartley-Kraus approach through a temporal opening with either an extradural or an intradural exposure of the sinus; (2) Mosher's anterior orbital approach with exenteration of the orbital contents and removal of part of the orbital plate; (3) the nasosphenoidal route of Langworthy; (4) the transantral route of Luc. A fifth method was advocated by Browder and Myerson,[12] viz., the coagulation of the most anterior arm of the cavernous sinus with the electrocoagulating current; this is to be applied only when infection having its origin in thrombophlebitis has entered through the ophthalmic veins, and must be done at the earliest possible stage.

REPORT OF A CASE

J. P., a white boy aged 9, was admitted to the pediatric service on May 29, 1939, with a history of swelling of the right side of the nose and face of four days' duration. He had chilly sensations, excessive thirst and restlessness with loss of appetite; he complained of feeling "bad." He also complained of general aching, with pain over the left side of the chest and in the knees. The swelling, once it started to spread, rapidly involved the entire right side of the face.

No history of any injury or accident could be obtained, and the mother stated that the child had been in good health all his life so far as she knew.

Examination showed the child to be very sick, with intense redness and edema of the right side of the face from the upper lip to the forehead. There were

12. Browder, J., and Myerson, M. C.: Surgical Method for Prevention of Thrombophlebitis of Cavernous Sinus: Report of a Case, Arch. Otolaryng. **21**: 574-583 (May) 1935.

several small pustules on the right cheek. The right eyelids were swollen and ecchymosed, and the eyeball was proptosed and immobile. The pupils were normal in size, shape and reaction to light. Ophthalmoscopic examination on the second day in the hospital showed choking of the disk amounting to about 4 diopters. There was a bloody purulent discharge from the right nostril. The teeth were in a poor state of preservation, with an ulcerated area about 1 cm. in diameter over the root of the right upper canine tooth.

The child was in a septic condition, with a temperature of 103 F., a respiratory rate of 40 and a pulse rate of 140. The left side of the face showed what appeared to be slight edema but no tenderness at that time. Pain at the time of admission required ¼ grain (0.016 Gm.) of codeine at two hour intervals for relief.

A smear of material from the ulcerated area on the gums the first day the child was in the hospital showed short chain streptococci and staphylococci as well as a few pneumococci. Urinalysis gave negative results. The blood count showed erythrocytes, 4,460,000, and leukocytes, 23,550, with 12 per cent lymphocytes, 3 per cent monocytes, 72 per cent neutrophils and 13 per cent stab cells.

Sulfanilamide was given (10 grains [0.6 Gm.] every four hours), and hot boric acid compresses were applied to the swollen area. The mouth was irrigated every four hours with an alkaline mouth wash, and fluids were forced. The nose was sprayed with a 25 per cent dilution of a solution of epinephrine hydrochloride (1:1,000).

On the second day of observation the left side of the face became swollen, the eyelids became ecchymosed and proptosis of the eyeball and paralysis of the third, fourth and sixth cranial nerves appeared, the left eye thus being affected exactly as the right eye had been on admission. Choking of the left disk was present two days later.

Repeated blood cultures gave negative results. Fever was of the septic type, the temperature varying from 100 to 103 F.

A transfusion of 250 cc. of whole blood was given on the third day, and this procedure was repeated every third day, from 150 to 250 cc. being given at each transfusion.

On the fourth day the child was cyanotic over the entire body, a condition which was thought to be due to the sulfanilamide. Methylthionine chloride was given, 10 grains (0.6 Gm.) every three hours, which greatly lessened the cyanosis. At this time the red cell count was 4,560,000 and the hemoglobin 75 per cent. The administration of methylthionine chloride was discontinued after twenty-four hours, owing to the lessening of the cyanosis.

Boric acid ointment and hot moist boric acid compresses were kept on the eyes to protect the cornea while the proptosis was extensive enough to prevent closure of lids. No ulceration formed on either cornea, but there was slight keratosis in both eyes.

The chemotherapy was discontinued on the sixteenth day, at which time the patient was allowed to sit up in a chair for a short interval. In all, 860 grains (55.5 Gm.) of sulfanilamide had been given.

The stormiest part of the stay in the hospital was the first four days, during which the child was constantly restless and irrational. At no time did he have any of the symptoms of meningitis.

Roentgen examination of the nasal air sinuses revealed no abnormality.

Treatment was conservative throughout on the basis of inability to find bacteria in the blood stream, causing the belief that the infection was probably imprisoned in the thrombus.

The spinal fluid was found to be normal except for increased pressure.

No evidence of septic emboli in any part of the body was found during the illness. The blood count varied, as shown in the following tabulation:

Date	White Cells	Red Cells	Hemoglobin	Stab Cells	Neutrophils
5/29	23,550	4,460,000	78%	13	72
6/ 9	23,700	3,280,000	70%	2	60
6/13	14,500	2,290,000	45%	2	63
6/17	11,400	3,270,000	60%	6	60
6/20	11,000	3,270,000	50%	4	56
6/26	11,300	4,110,000	75%	3	60

Results of general physical examination were negative at all times, with the exceptions already mentioned.

The child was discharged on the twenty-ninth day after his admission to the hospital. At this time the swelling of the face was entirely gone. The eyelids were normal in appearance and had normal motility. The eyeballs moved in the normal manner, and the corneas were practically clear. Only a small amount of haziness could be seen with the unaided eye. The pupils were round and regular and reacted to light and accomodation. Vision in each eye was 20/30. The ulcerated area on the right gum was entirely healed without scar tissue. The reflexes were normal. The mentality was normal. The choking of the disk had entirely disappeared. The spinal fluid was under normal pressure. The child was generally undernourished, weighing 55 pounds (24.9 Kg.).

After seven months an examination showed the child to be normal in every respect; vision was 20/15 in each eye.

Comment.—Judging from the temperature and the septic appearance of the patient, as well as by the blood picture, I am convinced that this was septic thrombosis of the cavernous sinus. Unless there was a complete walling off of the infected area by venous thrombosis, I am unable to explain why the infecting organism was never found on repeated blood cultures.

RECOVERIES RECORDED IN THE LITERATURE

Seydell [13] reported a case of thrombosis of a cavernous sinus in a young woman aged 19. The disease began in the ear, material from which showed hemolytic streptococci on smear. A radical mastoidectomy was done on the fifteenth day. Sulfanilamide amounting to 144.5 Gm. was given over a thirty-six day hospitalization, and the patient recovered.

Segar and Stoeffler [14] reported a case of mastoiditis and thrombosis of the lateral sinus in an infant 8 weeks old. Operation showed a mastoid full of creamy pus. The cavernous sinus when opened was not only thrombosed but frankly purulent. Staphylococcus was isolated. The patient recovered.

13. Seydell, E. M.: Recovery from Thrombosis of the Cavernous Sinus, Arch. Otolaryng. **30**:429-430 (Sept.) 1939.

14. Segar, E. H., and Stoeffler, W.: Mastoiditis and Lateral Sinus Thrombosis in an Infant, J. A. M. A. **96**:678 (Feb. 28) 1931.

Barnshaw [15] reported the case of a girl 7 years of age who had a sty on the eyelid three days previous to examination. She had squeezed it with her fingers. A diagnosis of bilateral cavernous sinus thrombosis was made. Ten cubic centimeters of prontosil was given every four hours for six doses. Sulfanilamide, 10 grains (0.6 Gm.), was given four times daily for four days, with recovery.

Browder [16] reported a case in which the disease started from a carbuncle on the right side of the nose. The carbuncle had come from a small pimple. The right eye showed typical signs of thrombosis of the cavernous sinus. Culture of pus from the carbuncle showed Staphylococcus. The same organism was grown on culture of the blood. The spinal fluid showed 20 cells per cubic millimeter. Treatment was directed toward occlusion of the cavernous sinus by electrocoagulation.

Through a right cranial opening, the right cavernous sinus was exposed by elevating the right temporal lobe. With the Bovil electrocoagulation unit, the sinus was completely obliterated up to the orbital fissure; the cranial wound was closed without drainage. The angular vein was opened, and an abscessed cavity which extended into the interior aspect of the orbit was disclosed. Drainage continued for about two months. There was complete recovery except for total blindness in the right eye.

Dupuy [17] reported a case in which the operation consisted of ligation of the internal carotid artery on the affected side. External canthotomy was done, followed by removal of the eye and of the contents of the orbital cavity. Considerable pus was encountered in the deeper area of the orbital cavity, posterior to Tenon's capsule. The lamina of bone between the optic foramen and the sphenoid fissure was removed with a chisel and a large curet, and an entrance into the cavernous sinus was made with blunt Mayo scissors. Little, if any, hemorrhage was noted, owing to blockage of the sinus by thrombus. A gauze drain was inserted in the sinus cavity. Several administrations of antistreptococcus and erysipelas antitoxins were given. Gentian violet and mercurochrome were given intravenously, as well as three blood transfusions. The patient recovered after three weeks.

Various operations on the sinus by the temporal route were discussed by Dwight and Germain [18] with a report of 4 cases in which recovery was achieved, including 1 in which cranial operation was done.

Faulkner [9] said:

> The percentage of recovery . . . is very small but has been placed as high as 7 per cent in Dr. Dorland Smith's series. The majority of these cases were of

15. Barnshaw, H. D.: J. M. Soc. New Jersey **36**:22-24, 1939.
16. Browder, E. J.: Laryngoscope **43**:829-830, 1933.
17. Dupuy, H.; Brewster, H. F., and Thomas, A.: Laryngoscope **42**:931-933, 1932.
18. Dwight, E. W., and Germain, H. H.: Boston M. & S. J. **146**:456, 1902.

aural origin. There are few recoveries from the anterior infection though one is reported in Dr. Eagleton's series. Recoveries in cases due to intranasal infection have been reported, but are very rare and there is some suspicion that some may have been an orbital abscess. The throat and neck cases have the worst prognosis of all, especially if the thrombus extends through the pterygoid plexus. Cases with extension through the jugular offer some promise from early resection of the jugular and lateral sinus.

Bowers [10] stated:

Among all the cases of cavernous sinus thrombosis from all causes only one has recovered as a result of operation on the sinus; about fifteen have recovered spontaneously. This would seem to indicate that the conservative treatment was best.

Eagleton advocated ligation of the carotid artery to give rest to the parts. He said that he had rarely seen bad effects from this operation.

Chisolm and Watkins [4] concluded that the approach should be determined by the location of the focus of infection. For instance, involvement of ethmoid or sphenoid cells would suggest the ethmoidal-sphenoidal route of Longworth; with orbital infections Mosher's orbital route could be used. Surgical failures undoubtedly are due to spread of the infection to surrounding sinuses and to the meninges.

MacNeal and Cavallo [19] stated that in recent medical literature there have been increasingly frequent reports of the successful care of patients afflicted with infections formerly regarded as almost certainly fatal. They said that so far as they were aware, however, recovery of a patient with bacteremia accompanied by obstruction in the cavernous sinus still remains a medical rarity.

Cavenagh [20] was able to find only 7 reported recoveries from acute thrombosis of the cavernous sinus. Three of the patients recovered after surgical drainage of the sinus, and only 1 of them had a positive blood culture (Staph. aureus). Four patients recovered after treatment directed against their infection. In 3 of these the disease was due to the staphylococcus, and this organism was recovered from the blood of 2. One of these patients with bacteremia received only transfusions; the other 2 were treated with bacteriophage. The fourth patient in this group given anti-infection therapy appears not to have had a positive blood culture. He was given a polyvalent antistreptococcus serum intravenously. Cavenagh found also a record of spontaneous recovery in an African native, without bacteriologic study or specific treatment.

19. MacNeal, W. J., and Cavallo, M. E.: Streptococcic Bacteremia and Apparent Thrombosis of Cavernous Sinuses with Recovery, J. A. M. A. **109**: 2139-2141 (Dec. 25) 1937.

20. Cavenagh, J. B.: Brit. M. J. **1**:1195-1199, 1936.

Grove [21] concluded that with the septic type in which the thrombosis or thrombophlebitis reaches the cavernous sinus by way of its afferent vessels the mortality is practically 100 per cent.

MacNeal and Cavallo,[19] in reporting on a patient who recovered, stated that they were not offering critical evidence to aid in deciding an argument about the therapeutic efficiency of the sulfanilamides or streptococcic serum or streptococcic bacteriophage; they disclaimed any immediate interest in exploiting any of these agents but hoped rather to bring some help and encouragement to physicians who are confronted with patients of this type.

CONCLUSION

On the assumption that this thrombus was walled off from the surrounding tissue and the general circulation one may conclude that the outcome would probably have been as favorable without sulfanilamide as with it. However, there may have been a few septic thrombi in the blood stream that were not detected by blood cultures. It is possible that the sulfanilamide might have placed the balance of recovery away from the fatal side. Undoubtedly, in view of all the favorable reports on chemotherapy in the form of the administration of sulfanilamide and considering the relative infrequency of severe reactions, the drug should be used in all cases of sinusal thrombosis.

In reviewing the literature I find that almost every reported cure was based on the prevailing treatment for septicemia at that particular time. Chemotherapy in the form of the use of mercurochrome or gentian violet was given credit for cures in 1932, and recoveries were attributed to electrocoagulation of the sinus, administration of antistreptococcus serum, ligation of the internal carotid artery and many other therapeutic methods up to the time when the use of sulfanilamide was introduced. A more careful study of those "cures," however, will reveal the fact that they occur only in isolated cases. Careful study will also reveal that in practically none of these cases was there evidence of septicemia or involvement of surrounding tissue in the form of meningitis.

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21. Grove, W. E.: Septic and Aseptic Types of Thrombosis of the Cavernous Sinus, Arch. Otolaryng. **24**:29-50 (July) 1936.

TUBERCULOSIS OF THE MIDDLE EAR AND MASTOID

MERVIN C. MYERSON, M.D.
NEW YORK
AND
JOSEPH G. GILBERT, M.D.
BROOKLYN

Knowledge of invasion of the temporal bone by the tubercle bacillus has increased greatly in recent years. Few of the earlier textbooks on otology mentioned aural tuberculosis. References to it were usually buried in the chapter on chronic suppurations of the ear. Louis mentioned that Ménière was the first to direct attention to the destructive action of the tubercle bacillus on the tympanic membrane. This, he claimed, resulted in deafness. Ménière's observations are of interest because they were made before the discovery of the tubercle bacillus by Koch in 1882. It was during the same year that Politzer first reported his histopathologic studies of tuberculosis.

In evaluating reports on tuberculosis of the middle ear and mastoid it is important to consider the age of the patient and the country from which the report emanates. There occurs a relatively large incidence of bovine tuberculous infection of the middle ear in countries where the milk is not pasteurized. The reported frequency of this condition varies with different authors. Leegard operated on 199 children under 10 years of age with suppurative otitis media. According to laboratory studies, 20 were tuberculous. Cox and Dwyer were able to isolate the tubercle bacillus in 15.6 per cent of the cases of ear infection observed by them among children by employing a special bacteriologic technic. Two per cent of 1,500 children examined by Westmacott were found to have tuberculous otitis media. Turner and Fraser found positive indications of tuberculosis in 1.8 per cent of 3,313 cases of otitis media. In their second group, of 4,285 cases, reported by McCart, 1.3 per cent were tuberculous. In the first series 9 adults and 51 children were affected, while in the second there were 46 adults and 9 children. Ninety-two per cent of the children in the first series and 50 per cent in the second were under 2 years of age. It was the opinion of Turner

From the Department of Otolaryngology, Sea View Hospital, Staten Island, N. Y.

and Fraser that more careful bacteriologic and histologic studies of the chronic suppurations of very young children would reveal a greater incidence of aural tuberculosis than that which they found.

Milligan expressed the opinion that this disease occurred much more frequently than was generally supposed. Spencer felt that the condition was overlooked, even among patients who were known to be tuberculous. This, he believed, was due to the absence of pain and the difficulty of isolating the specific bacillus from the discharge from the ear. Omerod encountered a 1 per cent involvement of the middle ear in tuberculous persons. Mathews found 7 instances of involvement of the temporal

TABLE 1.—*Otitis Media Complicating Pulmonary Tuberculosis*

Age of Patients	Cases
1 to 10 years	7
11 to 20 years	14
21 to 30 years	26
31 to 40 years	21
41 to 50 years	10
51 to 60 years	2
Total	80

TABLE 2.—*Otitis Media Yielding Tubercle Bacilli on Smear*

Age of Patients	Cases
1 to 10 years	1
11 to 20 years	2
21 to 30 years	6
31 to 40 years	11
41 to 50 years	4
51 to 60 years	0
Total	24

bone among 25 autopsies. Two thousand, five hundred and forty-one patients of adult age, residing in a sanatorium, were studied by Thomson. In 0.43 per cent there was evidence of specific infection of the middle ear. In a second group, of 766 adults, the incidence of infection was 1.82 per cent. Of the 1,650 patients in Sea View Hospital who were known to have tuberculous disease, 1.5 per cent had proved tuberculous otitis media.

It is generally conceded that many of the aural discharges among tuberculous persons are caused by the action of tubercle bacilli. In a fair percentage of such cases the bacilli cannot be isolated by any laboratory method at present available. Repeated examinations, however, will frequently establish the presence of these organisms. Tubercle bacilli were proved to exist in 35 per cent of the ears which we studied.

Among the 28 patients whose aural disease was considered tuberculous, the results of the laboratory examinations were as follows:

Cases	Smear	Culture	Biopsy
24	positive	not done	not done
1	negative	positive	not done
3	negative	negative	positive

When a smear showing the tubercle bacillus was obtained it was unnecessary to resort to culture or biopsy. Of 6 cases in which smears did not show the bacillus, in half the discharge was found positive on culture and in the other half the bacillus was found at biopsy.

Tuberculous infection of the middle ear may occur in one of two ways. According to some, the infected material passes into the middle ear by way of the eustachian tube during such acts as coughing, sneezing or regurgitation. This is more likely to occur in children, because their eustachian tube is shorter, more nearly horizontal and more patent than that of the adult. The mucosa of the eustachian tube may become directly involved by extension of tuberculous disease from the nasopharynx. Of interest is the fact that Milligan found evidence of tuberculous infection of the adenoid tissue in 15 per cent of the children he examined.

The second pathway of infection is considered to be the blood stream. Spencer, Dufays and Omerod considered this the more likely pathway. Retrouvez directed attention to the fact that involvement of the ear, bones, joints and other structures occurred simultaneously in tuberculous subjects. It was Omerod's opinion that in many cases tuberculous invasion of the mastoid occurred by way of the blood stream, from infected tonsils or lymphatic glands; he also felt, however, that in some cases the infection was due to the passage of bacilli through the eustachian tube after the ingestion of contaminated milk.

Infection of the middle ear and mastoid is either primary or secondary. The secondary form usually complicates glandular or pulmonary tuberculosis. Dufays and Goldstein have described cases of primary tuberculous otitis media and mastoiditis in children. Milligan felt that this condition was not rare; he considered it especially common among infants and children. Omerod was of the same opinion; he averred that the secondary form of the disease almost invariably attacked adults. Urbantschitsch observed clinical primary tuberculosis of the middle ear only in persons of advanced age. Some of the older writers considered the infection to be primary when it was characterized by otitis media with early involvement of the periauricular glands, enlargement of the adjacent cervical lymph nodes, occasional paralysis of the facial muscles and sequestration of parts of the temporal bone.

Most reports of cases deal with the secondary type of infection. Our experience would indicate that the primary form is extremely uncommon. We have seen 80 patients with aural disease during the past eight years, of whom 24 suffered with proved tuberculous otitis media. The number of new cases available for our study is restricted because of the long period which the average patient spends in our hospital.

Those who discuss primary tuberculous otitis media seem to overlook the presence of extrapulmonary foci. It is also significant that many of the reports of primary infection of the ear were made at a time when roentgen studies were not available. In previous years nonpasteurized milk was a prominent factor in the production of tuberculous otitis media in infants.

This condition is distinguished from the usual otitic infection because it is characterized by a symptomless onset and frequently a symptomless course. Such symptoms as pain, fever, headache and restlessness were absent in 92 per cent of the patients of Turner and Fraser at the first examination. In 88 per cent of the cases the parent became aware of the aural disease by discovery of the discharge. There was no other symptom than a discharge from the aural canal in 70 per cent of our cases. An elevation of the body temperature occurs only when a mixed infection exists. Loss of hearing is usually present early and becomes progressively worse. Swelling of the periauricular glands may also occur early. Facial paralysis occurs more frequently in the tuberculous form of chronic suppurative otitis media. This symptom was encountered by Turner and Frazer in 45 per cent of their cases. Millgan found an incidence of 8 per cent, while Leegard and Thomson did not encounter this condition. Omerod observed facial paralysis in a single patient among 26 with otitis media. We have seen facial paralysis in 4 of our 80 patients. Evidence of labyrinthine irritation in the form of dizziness and spontaneous nystagmus is occasionally encountered.

The discharge is usually not characteristic. In 70 per cent of our cases a serous discharge appeared first, to be replaced later by a purulent and fetid one. Diminished acuity of hearing, which in adults is associated with tinnitus, occurs early in the disease and becomes progressively worse. Cervical adenitis, which is mentioned by most authors, was not encountered in our study.

The first indication of the tuberculous nature of chronic otitis media or mastoiditis may be a slightly tender pale preauricular swelling, followed by enlargement of the postauricular glands.

The idea that multiple perforations of the tympanic membrane are frequently present in this disease has proved to be erroneous. All but 2 of our patients had a single perforation or total destruction of the

membrane. The 2 exceptions had double perforations. Leegard observed multiple perforations only once in 25 cases, Thomson twice in the same number and Omerod eight times among 17 cases. Leegard explained the variation of his figures from those of others by the difference in the material investigated. He maintained that there was a greater frequency of multiple perforations among adults in the advanced stage of pulmonary tuberculosis or infants and small children suffering from a fatal form of the disease. Most of his patients were in fairly good health. A majority of our patients, however, suffered from advanced bilateral pulmonary disease, yet none of them had multiple perforations of the tympanic membrane. When large perforations exist, granulation tissue is frequently seen protruding through them. This was the case in 4 of our patients.

TABLE 3.—*Analysis of Signs and Symptoms in Eighty Patients with Tuberculosis of the Middle Ear and Mastoid*

	Cases
Onset	
Acute	5
Insidious	75
Nature of discharge on first examination	
Serous	5
Seropurulent	20
Purulent	55
Symptoms	
Subjective	
Headache	
Dizziness	
Tinnitus	
Muffled hearing	
Objective	
Nystagmus	
Granulations	
Perforations of membrane	
Single	78
Multiple	2

Fifty-one patients appeared to have tuberculous otitis media, although not proved to have it; the 24 who had positive smears were among those in whom the onset was insidious.

Occasionally the cortex is perforated, revealing a sinus which leads to a cavity filled with caseous material. Such a condition was encountered in 3 of our cases; in each a sequestrum was found. Caseous material was found in the mastoid cavity or the middle ear in a majority of the patients who came to operation. In some the entire mastoid process was necrosed, so that large areas of dura were exposed. It is not uncommon to see a sequestration of part of the wall of the canal in cases in which a simple mastoidectomy has been performed previously. In such cases nature performs a modified radical operation. In two instances in which operation had not been performed before, the posterior wall of the canal was destroyed to such an extent that the cavity encountered resembled that seen after a modified radical operation. Paralysis

of the facial muscles may result when sequestration of bone which includes the facial canal occurs. The ossicles are frequently found lying free. The stapes has been found in the orifice of the eustachian tube and the malleus on the floor of the middle ear. In two instances the incus and malleus were extruded with the discharge. Sequestration of various parts of the internal ear occasionally occurs. Fraser reported a spontaneous cure of tuberculous labyrinthitis, which had occurred as a result of extensive sequestration.

There is no fixed rule concerning the pathway which a tuberculous infection of the middle ear will travel. The infection frequently passes through the round window to involve the cochlea, or it may attack the semicircular canals and cause fistulas of their walls. Meningitis resulting from this type of extension is not uncommon, according to Crowe. He expressed the opinion that the secondary tympanic membrane appears to be more resistant to the usual type of infection than it is to tuberculosis. It is extension through this region which accounts for the early deafness frequently observed in persons with this disease. Fistulas were found in the horizontal semicircular canal twice in this series. In each instance the membranous labyrinth remained intact.

At times the disease extends into the petrous portion of the temporal bone. It may also involve the meninges and the venous sinuses. Crowe, Mathews and Milligan expressed the belief that there is a direct extension from the middle ear to the meninges, whereas Goerke, Thomson and Fraser considered this to be rare. Our experience leads us to support the opinion of Fraser, that the dura is a much better barrier than the bone against tuberculous disease.

The sigmoid sinus and the internal carotid artery are attacked infrequently; only a few such cases have been reported. Grossman encountered a caseous fistulous tract leading to the sigmoid sinus. Extension of the disease to the internal carotid artery may cause severe and fatal hemorrhage. Wiening cared for a child less than 2 years of age, who suffered repeated arterial hemorrhages from the middle ear. Despite his operation, the patient died. The temporal bone was sectioned and revealed destruction of the carotid canal, with invasion of the internal carotid artery by tuberculous granulation tissue. One of our patients experienced sudden severe hemorrhages from a postauricular fistula. These hemorrhages were arterial, and repeated firm packing was required for their control. Numerous transfusions were necessary to replenish the blood. The hemorrhage was permanently stopped by resorting to an electrocoagulating instrument (case 8).

The diagnosis of tuberculous otitis media and mastoiditis depends principally on clinical and laboratory studies. A spontaneous serous or seropurulent discharge without pain, with only slight fever, with early

progressive deafness and enlargement of the periauricular glands and with or without facial paralysis and labyrinthine symptoms bespeaks the presence of a tuberculous process. Chronic suppurative infection of the middle ear occurring in the presence of pulmonary tuberculosis must be regarded with suspicion. However, a chronic discharge from the ear of a patient having pulmonary tuberculosis does not necessarily indicate a tuberculous basis. It may be extremely difficult to establish the specific nature of such a discharge, even though clinical observation leads one to suspect that it is tuberculous. We were unable to prove the presence of a tubercle bacillus by repeated smears, cultures and biopsies in 54 of our cases. At operation the tuberculous nature was recognized because of the presence of sequestrums surrounded by caseous material.

The recovery of the tubercle bacillus from the exudate of the middle ear or from the mastoid cavity at operation is conclusive, although Dean asserted that the isolation of this organism from the aural discharge is not positive evidence of tuberculosis. It is our feeling, however, that the finding of the bacillus in a smear obtained from the discharge of a patient with chronic otitis media associated with pulmonary tuberculosis establishes the diagnosis of tuberculosis of the ear. In many instances the tubercle bacillus is not found in repeated smears of the aural discharge. When such is the case, studies by means of culture mediums may be of value. At times it is impossible to isolate the organism by smear, culture or biopsy. When this is true inoculation of guinea pigs with material taken from the aural discharge or from the adjacent glands may be of value.

Occasionally a mastoid operation is performed in the belief that an ordinary suppurative lesion exists. The wound is slow to heal or never fully heals, leaving a discharging sinus. This should lead one to suspect an underlying tuberculous basis. Histologic study of sequestrums from the diseased area frequently yields evidence of the true nature of the disease.

A careful technic for isolating the tubercle bacillus is important; that evolved by Cox and Dwyer is good. Their technic is as follows: A solution of 3 per cent sodium hydrate is used to wash the discharge from the ear or mastoid wound. The discharge is then allowed to stand in a sterile flask in an incubator for from one to three hours. It is shaken at intervals. After the flakes of mucus have been dissolved, the mixture is thoroughly centrifuged, and the supernatant fluid is poured off. The precipitate is neutralized with normal hydrochloric acid, litmus being used as an indicator. The residue is now injected into a series of tubes containing Miller's culture medium. This medium contains an extract of glands and spleens of laboratory animals. It is used because of the well known preference of the tubercle bacillus for glandular tissue.

When tubercle bacilli are present, growth takes place in from ten to twenty days—much more rapidly than when the original Petroff medium is employed.

We have observed a thin serous discharge coming through the drum membrane, which, after the membrane is thoroughly wiped, is followed by a thick exudate. The thinner material is much more likely to yield tubercle bacilli on smear.

Roentgen study of the temporal bone may be of value. According to Law, a thin petrous bone suggests the possibility of tuberculosis. Besides the usual mastoid studies, roentgenograms should be made of the petrous pyramid. The latter are likely to reveal the presence of tuberculous disease around the labyrinth or in the deeper structures.

TREATMENT

The treatment is either operative or nonoperative. The nonoperative treatment consists of cleansing, using various medications in the form of powders and drops and light therapy. Direct exposure to the rays of the sun has been tried by some. Others have used a modified laryngoscope for indirect exposure.

There are various conditions which indicate the necessity for surgical intervention. Operation is performed when one or more of the following conditions are present:

Subperiosteal abscess.

Circumscribed labyrinthitis.

Facial paralysis.

Persistent postauricular fistula, after a simple mastoidectomy for tuberculous infection.

Mastoid tenderness and headache, associated with profuse discharge.

Opinions vary as to the indications for operation. Politzer operated on his patients when pulmonary tuberculosis developed in the presence of a long-standing suppuration of the middle ear. He was not inclined to operate when the aural condition occurred in the course of the pulmonary disease. Milligan reserved his operations for those cases in which the disease was not too far advanced and the general health not too bad. He felt that a dangerous focus of infection was removed by the operation. Ruttin operated only on patients who suffered severe pain. It is a fact, however, that most patients with tuberculous mastoiditis do not have severe pain. Muskat performed radical mastoidectomy on adults with aural and active pulmonary disease when he felt there was a drain on the patient's vitality. Graham insisted that early childhood is no contraindication to the performance of a radical mastoidectomy. This is in accord with our experience. In the treatment of their adult

patients Miller and Thomson never found an indication for surgical intervention. One must regard the absence of indications for surgical treatment among a large group of adults as a pure coincidence.

Contraindications for Operative Treatment.—The presence of tuberculous meningitis or of a generalized miliary type of infection is a definite contraindication to surgical procedure. When symptoms of meningeal irritation develop in a patient who has a chronic aural discharge, the possibility of a tuberculous infection must be considered. Patients who are greatly debilitated and may not be able to withstand the shock and strain of operation should not be subjected to it.

Operative Treatment.—Accumulated experience has demonstrated that the best results are obtained by resorting to the radical mastoid operation. Radical surgical intervention has been advocated by Turner and Fraser, Bárány, Scobee, Muskat and others. Collet and Mayoux preferred the simple mastoidectomy. Analysis of their results, however, would indicate that the simple operation was inadequate, because there was a discharge either from the middle ear or from the mastoid wound for long periods after operation. Culbert performed a simple mastoidectomy on one of his patients; this did not result in healing. Subsequently he performed a radical operation, and complete healing resulted. A recent experience demonstrates the advantage of the radical procedure: A boy 3 years of age had a simple mastoidectomy on the left at another institution thirteen months before admission to Sea View Hospital. When he was first seen, a draining postauricular fistula was present; the discharge was thick and fetid. A radical mastoidectomy was performed by us on the right ear, because of a postauricular subperiosteal abscess which complicated a chronic infection of the ear. At the end of twelve days the postauricular wound had completely healed. The canal was dry and the cavity was completely epithelized at the end of eight weeks. After the radical operation there was marked improvement in the general health of the patient. On the one side radical mastoidectomy yielded a perfect result, while on the other a simple mastoidectomy left an aural infection, with a draining, fistulous condition of the tract. At the end of three years the right ear remains healed; the left ear continues to discharge posteriorly.

In a second case a simple mastoidectomy had been performed before the time of admission. A purulent discharge continued from the aural canal and from a postauricular sinus after admission of the patient to the hospital. A little later a sequestrum which proved to be tuberculous was extruded through the sinal tract; facial paralysis followed. A radical mastoidectomy was performed; this resulted in complete cessation of discharge and recovery from the paralysis. The patient's general health was greatly improved.

The choice of an anesthetic agent is important. Local anesthesia obtained by the infiltration of solution of procaine hydrochloride is preferred. This anesthetic agent can be used for all types of patients, regardless of age. Its use is facilitated by appropriate preliminary sedative medication.

REPORT OF CASES

CASE 1.—A colored boy aged 14 was admitted to the hospital July 26, 1937. At the time of admission a diagnosis of tuberculosis of the right malar bone, the tenth dorsal vertebra, the left tenth rib and one of the metatarsal bones of the right foot, together with tuberculosis of the hilar lymph nodes, was made. Multiple draining sinuses were present on the left side of the neck, in the lumbodorsal region and over the dorsum of the right foot. The patient was first seen by the otologist in January 1938, when he complained of pain and of discharge from the left ear. When the yellow purulent discharge was wiped from the aural canal, a reddish granulomatous mass was revealed. The mastoid tip was tender on pressure. A large, soft, fluctuant, nontender swelling was present in the left posterior cervical region. The hearing was impaired; whispered voice could not be heard; conversational voice was heard at a distance of 4 inches (10 cm.). The Weber test showed lateralization to the left side. The right middle ear and mastoid were apparently normal. Special examination indicated normal function of the labyrinth.

A simple mastoidectomy was performed on January 27, with the aid of local anesthesia. The simple operation was chosen because of the relatively short duration of the otitis media and because of the presence of pain, together with the absence of tubercle bacilli in the discharge. Pus was found under pressure in the mastoid cavity; necrotic bone was removed from the region of the horizontal and the posterior semicircular canals. The zygomatic cells were found involved. Material taken from the mastoid at operation revealed no evidence of tuberculosis.

Six days after the operation the granuloma became detached from the aural canal. Histologic examination showed large granular areas containing swollen fibrous bundles of tissue, numerous nuclear remnants and intact lymphocytes and polymorphonuclear leukocytes. Fibroblasts and epithelioid cells were seen in the periphery. Sections stained by the Ziehl-Neelsen method revealed tubercle bacilli.

The postauricular wound healed completely, but the patient died nine weeks after operation. Postmortem examination disclosed tuberculous meningitis and evidence of chronic hematogenous pulmonary and extrapulmonary disease. There was also involvement of all the viscera and of the spine. The patient succumbed to the effects of generalized miliary tuberculosis.

Examination of the skull revealed a bony excavation 1 centimeter in diameter, which occupied the inner plate of the temporal bone that had not been operated on. Greenish yellow pus, which proved to be tuberculous, was found on the adjacent dura and the pia-arachnoid. The temporal meningitis was caused by this focus, far removed from the site of the operation.

Comment.—This patient had acute otitis media, the symptoms of which continued for one month. Smears and cultures were repeatedly negative for the specific bacillus. The granuloma which was present in the aural canal proved to be tuberculous.

CASE 2.—A white man 22 years of age was admitted in June 1933, because of extensive bilateral caseous pneumonic tuberculosis, which persistently yielded evidence of tubercle bacilli in the sputum. A simple mastoidectomy had been performed on each side three months before admission, because of infection of the middle ear of three months' duration. When first seen, both postauricular wounds were draining, and there was a profuse discharge from each aural canal. Five months after admission a headache developed in the left temporal region, associated with dizziness and nystagmus to the same side. The hearing was completely destroyed. The vestibular reactions were normal. A sequestrum was extruded from the left postauricular wound. Smears and cultures did not establish the presence of tubercle bacilli. A radical mastoidectomy was performed.

Pus and granulation tissue, in addition to caseous material, were found in the middle ear and in the mastoid cavities. The headache disappeared, but the nystagmus continued. The wound began to break down twenty-seven days after operation, even though there was only scant discharge from the aural canal. Three days later a condition of general sepsis developed. The patient's pulmonary disease had extended, and his larynx became markedly edematous. On the following day he died.

Postmortem examination revealed involvement of the gastrointestinal and genitourinary tracts. There was also evidence of tuberculosis of the larynx and of both mastoid areas. Study of the granulation tissue and caseous material obtained during the mastoidectomy on the right disclosed the presence of tubercle bacilli.

Comment.—This case demonstrates the lack of value of a simple mastoidectomy for this disease. It also emphasizes the difficulty experienced in proving that an aural discharge is tuberculous.

CASE 3.—A 22 year old iron worker was admitted in July 1937. His tuberculous condition was recognized in May of the same year, when he complained of cough, night sweats, hemoptysis and loss of weight. He had a far advanced caseous cavernous involvement of the upper lobes of the lungs. In May 1938 he complained of muffling and clicking sensations in the left ear. This condition had lasted for several weeks, and during the past three days there had been a mucopurulent discharge from the aural canal. Slight pain was experienced. A few days later he complained of headache and pain in the left ear and jaw; in addition, he suffered from insomnia and occasional attacks of dizziness. Examination revealed thickening and redness of the ear drum, together with slight induration in front of the tragus and in the postauricular region. There was tenderness over the mastoid antrum and at the root of the zygoma; a horizontal nystagmus to the opposite side was now evident. The auditory function was depressed, so that whispered voice could be heard at a distance of 6 inches (15 cm.). Roentgen examination revealed absence of pneumatic structure but no evidence of destructive change. Five days later there was only slight mastoid tenderness and no tenderness in the zygomatic region. The nystagmus and headache had disappeared. On May 28 the discharge became more profuse and transmitted a pulsation. Two perforations of the membrane were noted, a granuloma being present in one of them.

On June 8 a radical mastoidectomy was performed. The mastoid process was sclerotic; the antrum was filled with thick pus and pale granulation tissue. This tissue proved to be tuberculous. The postauricular wound did not heal in its lower third. The patient's general condition became worse, and he succumbed on August 11.

Postmortem examination demonstrated that the patient had suffered from far advanced bilateral caseous pneumonic tuberculosis, with extensive cavitation. In addition, there was present tuberculous empyema and enteritis.

Comment.—Numerous smears from the aural discharge were negative, while material obtained at the time of operation proved the specific nature of the infection. The postauricular wound continued to discharge and failed to heal because of the severe general infection. It is questionable whether a patient suffering from such extensive disease should be subjected to mastoidectomy. The otologist must determine the advisability of operation and the prospect of a cure on the basis of the patient's general condition.

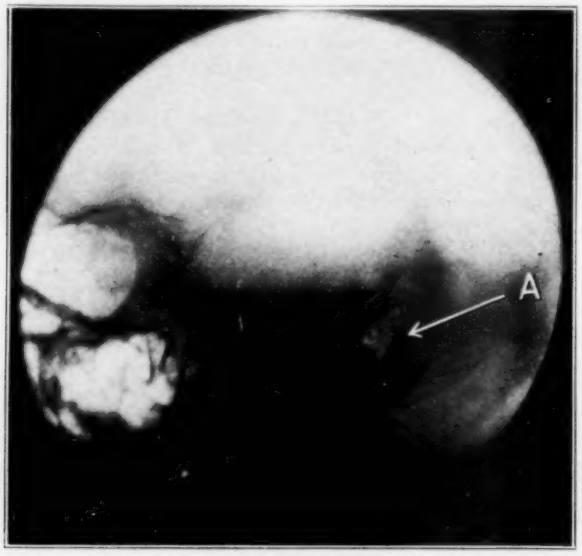

Fig. 1 (case 4).—Roentgenogram of mastoid regions. There is a large excavation (*a*) and a generally blurred appearance, indicative of extensive destruction and widespread disease.

Case 4.—A 25 year old Chinese laundry worker was admitted in May 1937. His tuberculous disease became evident in 1934 because of a swelling in the left side of his neck. Up to the time of his admission this swelling had been incised six times. There were now three draining sinuses in the cervical region and one in the left lumbar region. The lower lobe of his right lung was diseased, and the eleventh and twelfth ribs on the left side were involved in a destructive process. The left kidney was also affected.

The patient was referred for otologic study on September 14 because of diminished hearing in the left ear and occasional pain. Examination revealed congestion along the handle of the malleus and some retraction of the tympanic membrane. He was not seen again until January 9 of the following year, at which time a thin mucoid discharge was coming through an anterosuperior perforation of the ear drum. On February 6 a large granuloma was found protruding from this

perforation. It bled readily when touched. Repeated smears failed to show the tubercle bacillus. In June the discharge was proved to be tuberculous. Biopsy of the granuloma was reported by Dr. Auerbach as follows:. "Large and small pink granular areas are present, which indicate caseation. These contain nuclear remnants, intact lymphocytes and polymorphonuclear leukocytes. Epithelioid cells, lymphocytes, fibroblasts, collagen fibers and giant cells are present in the periphery. Diagnosis: tuberculous granulation tissue."

According to the roentgenograms, there was no destruction, but a reduced degree of aeration and a thickening of the cell walls. The ear was treated conservatively. In the latter part of November the skin over the tip of the mastoid area presented a bluish red swelling, which was painless and not associated with other symptoms. Roentgen study now showed definite destruction in the mastoid region, especially in the parts adjacent to the posterior canal wall. Caloric tests showed absence of the labyrinthine function.

A radical mastoidectomy was performed. Evidence of caseation was found in the middle ear and in the mastoid; this extended into the zygomatic region and the glenoid fossa. The postauricular wound healed completely at the end of a week, and epithelization progressed normally.

Comment.—There was a good response to the operation. This experience emphasizes again the difficulty encountered in obtaining the tubercle bacillus from the aural discharge. It demonstrates the importance of using all available methods to arrive at a positive diagnosis. This is the only case we have seen in which both the cochlear and the vestibular response were absent.

Case 5.—A 29 year old roofer was admitted Jan. 9, 1936. He had suffered in 1924 with severe bronchitis, which was subsequently diagnosed as tuberculosis. He had had an intermittent discharge from the left ear for the past ten years and was experiencing slight attacks of vertigo. The left drum membrane was thick and pale pink and exhibited a large posterosuperior perforation. There was no nystagmus. Roentgen study revealed an area of destruction just above the knee of the sigmoid sinus. Tubercle bacilli were present in smears made from the discharge. On March 16, in addition to the symptoms previously related, there was a rotary nystagmus on looking to the left. Spontaneous past pointing and falling were absent, but there were occasional attacks of dizziness, and the patient experienced a sensation of falling backward or forward. Whispered voice was heard only a few inches from the left ear. The Rinne test was positive for each side; the Weber test did not reveal lateralization. Caloric tests yielded essentially negative results.

A radical mastoidectomy was performed. The ossicles were missing; a large caseous mass filled the cavity of the middle ear. This tissue proved to be tuberculous. The wound healed nicely, and the postoperative course was uneventful.

Comment.—Extensive involvement, with symptoms of serious disease, was successfully eradicated by a radical mastoidectomy.

Case 6.—A colored boy 3½ years of age was admitted in October 1931. Before admission he had suffered with measles, bilateral acute otitis media and tuberculous cervical and peribronchial adenitis. A simple mastoidectomy had been performed on the left side in June. At the time of admission both ears were discharging

a thick yellowish exudate. The wound resulting from the mastoidectomy on the left had healed. In September 1932 a fluctuant swelling appeared over the right mastoid process.

A radical mastoidectomy was performed with the aid of local anesthesia. The cortex presented a perforation, beyond which lay a large sequestrum surrounded by caseous material. Granulations were found on the sigmoid sinus; the facial ridge had been destroyed to such an extent that the mastoid cavity gave the impression that a previous radical operation had been performed. The postauricular wound was completely healed at the end of sixteen days, and the wound cavity was dry within two months. The patient improved rapidly. Several years later a follow-up disclosed that the right ear was completely dry, while the left ear, on which a simple mastoidectomy had been performed, continued to drain.

Comment.—The superiority of radical mastoidectomy over the simple procedure is again demonstrated.

CASE 7.—A 23 year old white man was admitted in May 1933, because of bilateral caseous pneumonic tuberculosis. During the past three months the hearing in his right ear had been greatly impaired; during the past month the ear had been discharging. The left ear was similarly affected for a period of eight months. In August he complained of vertigo, nausea and right-sided headache. Otologic examination revealed a narrowed canal on the right, with a mucopurulent discharge coming from behind a polyp which almost completely occluded the entrance to the cavity of the middle ear. The hearing was gone. On the left side a mucopurulent discharge was coming through a large perforation in the drum membrane. Whispered voice could be heard 3 inches (7.5 cm.) away and conversation at a distance of 1 foot (30 cm.). Caloric tests revealed normal responses for the left ear and slightly delayed responses for the right. Spontaneous nystagmus directed to the right side was noticed. Cultures of the discharge from the right ear contained tubercle bacilli. Roentgen studies revealed evidence of destruction on the right side.

A radical mastoidectomy was performed. This disclosed marked necrosis of the mastoid area; a sequestrum approximately 1 cm. in diameter was found lying free in the antral region. The horizontal semicircular canal presented a fistula about 3 mm. long. The stapes was found lying in the cavity of the middle ear. Shortly after operation facial paralysis was noted; there was also nystagmus to both sides. Six days after operation the postauricular wound had healed. Three months after operation the wound was entirely dry, and the facial paralysis disappeared.

Comment.—This case represented involvement of the cochlea and the perilabyrinth. The infection most likely progressed through the oval window. The fistula in the horizontal canal led only into the perilabyrinth, the membranous labyrinth being intact.

CASE 8.—A white man 44 years of age was admitted in September 1933 because of chronic bilateral pulmonary involvement, with extensive cavitation. The results of examinations of his sputum were consistently positive. In April of the same year a simple mastoidectomy was performed on the right side, together with ligation of the internal jugular vein. Shortly thereafter the common carotid artery was ligated. During the next few months the mastoid wound was revised several times. Culture of the material from the mastoid region yielded Staphylococcus albus.

At the time of admission a large fistulous opening was present in the postauricular region; this led into the depths of the mastoid cavity. A large amount of granulation tissue surrounded the edge of the wound. A slight serous discharge was present in the right external canal. On several occasions smears were negative, but cultures revealed the presence of tubercle bacilli.

On April 12, 1934 a profuse hemorrhage occurred from the depths of the wound and was controlled by tight packing. The packing was removed twenty-four hours later. On April 24 there was another profuse hemorrhage, which was controlled by packing. The following day, on removal of the packing there was a recurrence of hemorrhage. The hemoglobin content of the blood dropped to 32 per cent and the number of red cells to 2,400,000. These hemorrhages recurred so frequently that on May 23, in spite of the patient's extensive pulmonary disease and general debility, an attempt was made to prevent future bleeding. A diathermic electrode was inserted into the anteroinferior region of the middle ear, in what was thought to be the location of the bend of the internal carotid artery. Bright red blood, accompanied by a definite pulsation, could be observed coming from this location. The electrode was kept in place for about one minute, until the bleeding was completely controlled. The patient died five months later as a result of extensive visceral changes. Postmortem examination revealed evidence of caseous necrosis of the right mastoid process and fibrous occlusion of the internal carotid artery at its knee, where it had been coagulated.

Comment.—The severe bleeding is of particular interest, as is the number of operations which were performed. During four and one-half months prior to the time of admission five operations were done. These included ligation of the common carotid artery and of the internal jugular vein. Recurrence of the repeated profuse hemorrhages from which the patient suffered was prevented by electrocoagulation of the knee of the internal carotid artery. Perhaps this severe complication might have been prevented, and the necessity for subsequent operations eliminated, by a well performed radical mastoidectomy in the first place.

Case 9.—A 6 year old colored boy was admitted to the hospital in May 1934. For two years before the time of admission he had had a recurrent discharge from the left ear. A month earlier a simple mastoidectomy had been performed because of a postauricular swelling. Material removed from the mastoid wound during the operation was found to contain tubercle bacilli. A few months after admission a sequestrum was removed from the mastoid wound; this proved to be tuberculous. The postauricular wound continued to discharge. Further surgical procedure was considered inadvisable because the patient was acutely ill. When, in June 1935, his condition seemed to be less acute, a radical mastoidectomy was performed. Facial paralysis of the left side had appeared a few weeks earlier.

At operation the dura was found exposed and adherent to the subcutaneous tissues. During the separation of the skin from the dura it was torn, and the tear was covered with a piece of temporal muscle. The cavities of the middle ear and mastoid were filled with granulations, and the horizontal portion of the facial nerve lay uncovered. The postoperative course was uneventful; at the end of two months the wound was completely healed and the cavity dry. By the following April the paralysis had completely disappeared.

Comment.—A simple mastoidectomy was not extensive enough to cure the tuberculous infection, for it left some residual disease and a draining fistulous tract. Facial paralysis finally made its appearance, and

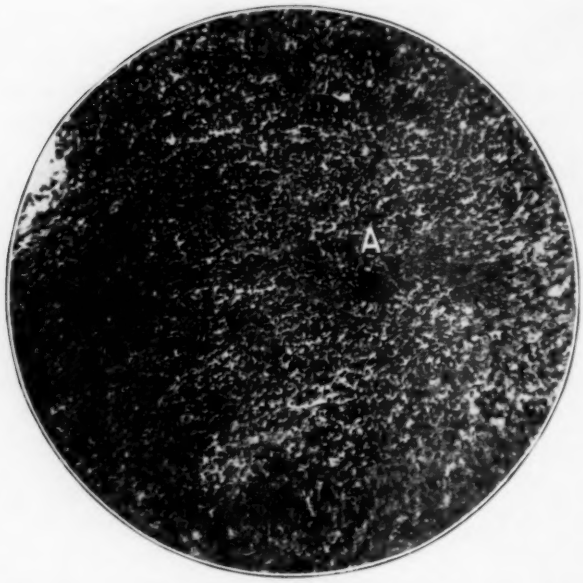

Fig. 2.—Photomicrograph of a specimen removed during mastoidectomy. This tuberculous granulation tissue is representative of the specimens removed from all tuberculous mastoid processes. A giant cell is marked *A*.

Fig. 3.—Photomicrograph of a section of ossicle found during operation. Note the tuberculous elements surrounding the bone.

the general condition of the patient was worse. Subsequently radical mastoidectomy eliminated the fistula, cured the facial paralysis and so improved the child's condition that he was able to return to school.

CASE 10.—A white man 40 years of age was admitted in January 1937. He had been coughing for four years and had lost some weight. His ears were free from disease at the time of admission, but two months later he noticed a watery discharge coming from the right aural canal. He had noticed a muffled sound in this ear for two weeks before the discharge made its appearance but had had no pain. When seen by the otologist, in June, examination disclosed a large central perforation, through which a moderately profuse discharge was coming; there was some slight tenderness of the mastoid tip. Two months later the patient

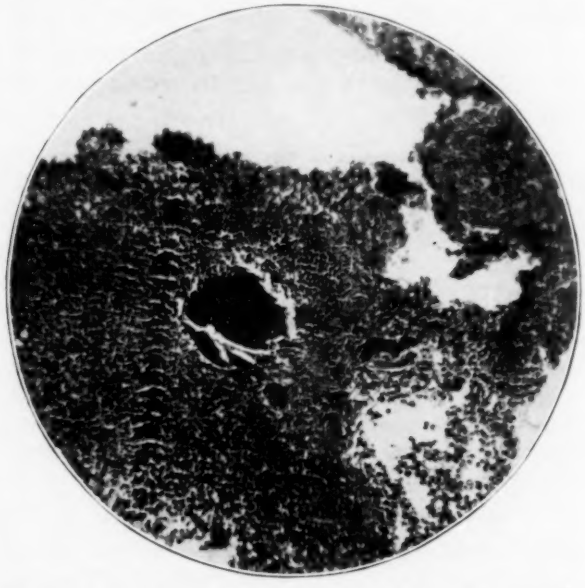

Fig. 4.—Photomicrograph of tissue removed at mastoid operation. Note the bone in the center of the field, surrounded by tuberculous elements. Tubercle bacilli were found when the section was stained by the Ziehl-Neelsen method.

complained of frequent attacks of dizziness and slight headache. The hearing was markedly diminished. Spontaneous horizontal nystagmus directed to the right side was noted, and caloric tests indicated that the labyrinths were functioning normally. In the latter part of August the patient complained of an almost constant headache in the right temporal region.

A radical mastoidectomy was contemplated, but on account of technical difficulties a simple mastoidectomy was performed. The lower half of the mastoid process was found to be replaced by a cavity filled with pus and granulation tissue. The sigmoid sinus was found uncovered in its entire length. Within three weeks the middle ear and the postauricular wound were dry. Several months later, however, a slight mucoid discharge could be seen coming through the large perforation. This discharge has continued up to the present time. Material taken from the mastoid area during operation was found to contain tubercle bacilli.

Comment.—Because of injury to the sinus the operator was forced to be satisfied with a simple mastoidectomy. If a radical operation had been performed, the middle ear would probably have remained dry.

CASE 11.—A 15 year old white girl was admitted in July 1938. She had been ill since the age of 7, when she had what was considered a tuberculous condition of the hip. In April 1939 a mucopurulent discharge made its appearance in the left aural canal; it was unaccompanied by pain or other symptoms. Examination revealed a bulging, reddened ear drum having an anterior perforation. Smears made from the discharge contained tubercle bacilli. The patient was treated conservatively until June 30, when she complained of severe pain in the left temporal region. A radical mastoidectomy was performed; the mastoid cells were found to be filled with a gelatinous serous material. The postoperative course was uneventful; the wound of the middle ear and mastoid healed completely.

Comment.—The occurrence of a discharge from the ear unassociated with pain in a tuberculous person should be considered as evidence of tuberculous otitis media.

CASE 12.—A white man 36 years of age was admitted in September 1939. A modified radical mastoidectomy had been performed in May. He now presented a discharging wound of the left mastoid; the middle ear also discharged. As a result, a revision of the mastoid wound was undertaken in August. It was at this time that evidence of pulmonary disease was discovered. At the time of admission the mastoid wound presented a gray, necrotic lining. The remnants of the malleus were found during the examination. Hearing on the involved side was almost completely lost.

Roentgen study revealed an irregular, moth-eaten appearance of the lateral and inferior borders of the mastoid cavity. There was a suggestion of erosion of the dural plate. Smears revealed tubercle bacilli. Operation was performed early in October and the wound was revised. The advanced pulmonary condition has prevented complete healing of the wound.

Comment.—The tuberculous disease made its first appearance in the form of a painless discharge from the ear. When the general condition is poor, complete recovery is not to be expected even after radical mastoidectomy.

CASE 13.—A colored woman 38 years of age was admitted in November 1938. Her illness dated from July 1936, when she complained of cough, expectoration, night sweats and weakness. Both ears had been discharging since July 1938. She was markedly deaf and had been hoarse since October. She was suffering with extensive bilateral caseous pneumonic tuberculosis. Her larynx presented partial destruction of the epiglottis, with some infiltration of the arytenoid cartilages and a granuloma in the interarytenoid space.

The right aural canal was filled with polypoid granulations, which bled when touched; a pulsating discharge was also present. On the other side a mass of polypoid tissue completely filled the inner part of the canal. Roentgen study revealed marked destruction of the left mastoid process. There was no such evidence on the right. Paralysis of the left side of the face appeared on March 2. For this reason a radical mastoidectomy was performed.

A large excavation, extending from the antrum half way to the mastoid tip and filled with caseous material, was discovered. The posterior canal wall was com-

pletely eroded, so that the facial nerve was exposed just below the horizontal semicircular canal. Destruction of bone led into the temporomandibular joint. All specimens contained tubercle bacilli. The facial paralysis disappeared within seventy-two hours after operation. The postoperative course was uneventful; the middle ear and the mastoid cavity healed nicely.

SUMMARY AND CONCLUSION

The cases of mastoiditis were accumulated during a period of seven years, while those of otitis media represent the number seen during a single year (1938). Our experiences demonstrate that a discharge from the middle ear appearing without pain in a tuberculous person should be considered as tuberculous.

The difficulty of making a positive diagnosis of the specific nature of the aural infection is emphasized. Resort should be had to smear, culture and biopsy, if necessary, to make the diagnosis.

A radical mastoidectomy with the region under local anesthesia should always be done, regardless of the patient's age, if the surgeon hopes to obtain a favorable result. The locally injected anesthetic may be supplemented if necessary by an intravenously injected anesthetic, one of those more frequently used for this purpose being chosen. There is no place for the simple mastoidectomy in the treatment of this disease, for the result is invariably a continuance of the pathologic process. When the pulmonary disease is greatly advanced and menacing the existence of the patient, surgical treatment of the ear should not be contemplated.

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DECUBITAL ULCERS OF THE PHARYNX

RICHARD WALDAPFEL, M.D.
GRAND JUNCTION, COLO.

Heryng [1] (1890) was the first to describe, under the title "Benign Ulcers of the Pharynx," a condition characterized by the following signs:

It occurs usually unilaterally, always in the shape of an oblong shallow ulceration or deep excoriation, and always on the anterior pillar over the tonsil. The margins are sharp, the floor in the beginning covered with a gray-white coating which can be removed by scraping. The surrounding mucous membrane is reddened. It does not bleed when touched and is rather indolent. It never originates from vesicles or pustules by confluence (Herpes, Pemphigus) and remains always solitary, and never are similar changes to be found on other places of the pharyngeal mucous membrane. After two to three days' duration red granulations appear on the floor of the ulceration. The papillary layer clears and from the edges the new epithelium proceeds toward the center, the coating disappears and after ten to twelve days epithelization is terminated without visible scar formation.

This description corresponds to the condition observed in case 1 of the following report, which recently came under my observation.

REPORT OF CASES

CASE 1.—Miss G. W., 27 years of age, came to the office on Nov. 29, 1939 complaining of a slight difficulty in swallowing, referable to the left side. At the same time there were tiredness, loss of appetite and a slight rise of temperature (99 F.). Examination of the pharynx showed the following picture (fig. 1): The left anterior pillar was bulged forward. It was reddened and showed on the greatest prominence of the swelling facing the tongue a sharply limited oval gray-white ulcer, which did not bleed when touched. The tonsils were not present, but in the uppermost part of the left tonsillar fossa, behind and under the ulcer, there was a remainder of tonsillar tissue, which was enlarged by inflammation. The ulcer was visible only when the tongue was depressed; otherwise it was covered by the tongue. The rest of the pharyngeal mucous membrane was normal, as were the right part of the soft palate and the uvula. Figure 1 shows distinctly the localization and extent of the ulcer and suggests also, as far as is possible in a black and white photograph, the difference in color between the inflamed left and the normal right side and the swelling of the left side. The condition had existed for two days. Injuries of any kind (trauma, scalding, burns) could be definitely disallowed. Under conservative treatment—gargling with hot fluids and touching with 5 per cent silver nitrate—the ulcer epithelized from the periphery and within eight days healed completely without leaving any scars.

Presented before the Colorado Otolaryngological Society, Denver, Jan. 6, 1940.

1. Heryng, T.: Ueber benigne Pharynxgeschwüre, Internat. klin. Rundschau 4:1681 and 1721, 1890.

This was a genuine benign ulcer of the pharynx, as described by Heryng. It occurred, strangely, in a patient without tonsils but had developed over a swollen remainder of tonsillar tissue in the anterior pillar.

Aphtha was considered as a possibility in differential diagnosis, but aphtha has a red halo, which was missing from the ulcer in this case, and almost never has an infiltrated underlying tissue such as that which was present in this case.

Heryng regarded this type of ulcer as very rare. He gave it, as he said, the name "benign pharynx ulcer" only provisionally, until the cause could be found. And in order to find the cause he performed inoculations on the anterior pillar of man with streptococci which he

Fig. 1 (case 1).—Photograph of the pharynx, showing *U*, the ulcer and *T*, the tongue partially depressed by a tongue depressor.

obtained from ulcerations such as that which he described.[1] These inoculations were unsuccessful. Freudenthal [2] said he regarded the condition as rheumatic, but he did not substantiate his belief.

In the last few years I have had occasion to examine and study further ulcerations of this type [3] which occurred under my observation, so that I could follow their development. This enabled me to draw conclusions concerning their pathology and etiology. Three of them will be reported in this paper. The shape and localization are shown in schematized sketches.

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2. Freudenthal, W.: On Rheumatic and Allied Affections of the Pharynx, Larynx, and Nose, M. Rec. **47**:196, 1895.

3. Waldapfel, R.: Ueber Kontaktgeschwüre im Pharynx, Monatschr. f. Ohrenh. **66**:1104, 1932.

The ulceration in case 2 (fig. 2*A*) corresponds with respect to localization and size with that in case 1, but the other findings are quite different.

CASE 2.—A melanosarcoma of the left tonsil became much larger soon after roentgen treatment and bulged the left faucial pillar forward, so that it touched the left lingual tonsil. The ulceration had developed under observation and within one day after the enlargement of the tonsil. It was visible only when the tongue was depressed, for it lay on the anterior pillar exactly where the latter touched the tongue. The extent of the ulceration corresponded to the contacting surface between both bulgings of the base of the tongue and the anterior pillar. (Roentgen reaction and ulceration receded within eight days.)

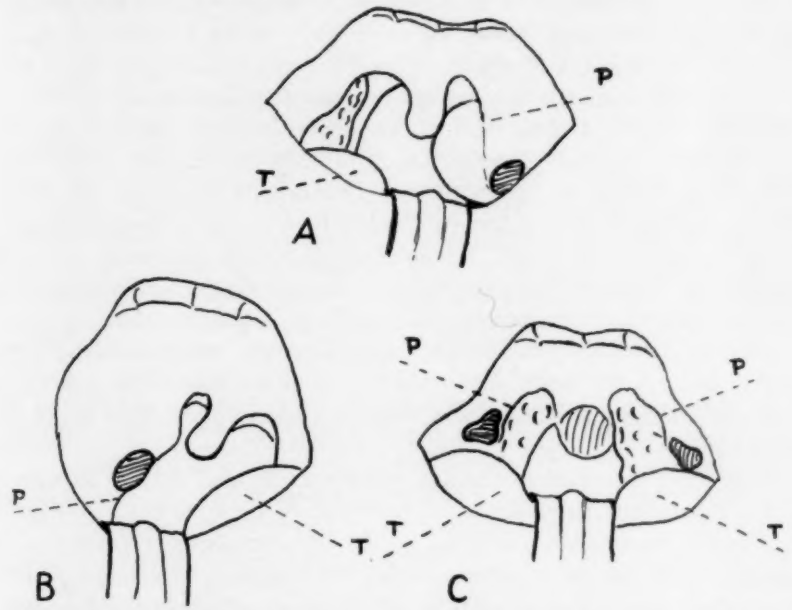

Fig. 2.—Schematic sketches: *A*, case 2; *B*, case 3, and *C*, case 4. *P* indicates the anterior pillar, *T*, the tongue, and the shaded area, the ulcer.

CASE 3.—An ulceration similar to that in case 2 developed on the right anterior pillar on the basis of a peritonsillar abscess, also exactly on the place where the base of the tongue touched the bulging anterior pillar. The ulceration developed one day after the formation of the peritonsillar abscess. Its shape and extent are adequately shown by the sketch (fig. 2*B*).

CASE 4.—In a case of high grade lacunar tonsillitis, in which there was an edematous swelling of both anterior pillars and of the uvula, under my observation there developed in the lower section of both anterior pillars a sharply limited ulcer with a gray-white coating (fig. 2*C*). On the uvula there was a not so deep but sharply limited epithelial erosion. When the mouth was simply opened and the tongue was resting, these changes were invisible; they were covered by the base of the tongue. If one depressed the tongue slowly with a tongue depressor, it became clear that the tongue had touched the pillars and the uvula exactly on

the ulcerated areas. The three ulcerated spots represented an archlike line which corresponded definitely with the line of contact of the most prominent parts of the edematous swellings with the tongue.

On the basis of these observations, I have explained the mechanics of the development of ulcers such as these as follows: The ulcer develops where the opposite bulgings of the tonsillar swellings and the base of the tongue continuously press the mucous membrane of the anterior pillar, which lies between them. If the uvula is much swollen, such an ulcer may exceptionally develop also on the uvula, as case 4 shows.

There can be no doubt, in view of these findings, that in the cases described here there was a decubitus formation of mucous surfaces facing each other and contacting each other under a certain pressure. Essential for the development of these ulcers, therefore, is a swelling of the tonsillar area which brings the opposite mucous membranes of the anterior pillar and the base of the tongue in prolonged contact. The pressure of contact produces a "decubitus" on the anterior pillar. The swelling itself may be caused in one instance by tonsillitis, in another by a peritonsillar abscess and in another by a tumor with reaction after roentgen treatment. But the swelling is always primary, the ulcer secondary. Because the ulcer outlasts the swelling, in later stages frequently only the ulcer, and not the swelling, is seen.

Recently Miyakogawa [4] has reported cases such as these. His explanation of the genesis of the ulcers follows mine. In one of his cases the swelling was caused by tonsillitis and in another by a peritonsillar abscess.

The developmental mechanics explain easily and readily all clinical and characteristic signs of the benign ulcer of the pharynx: the regular appearance in typical places—i. e., on the contacting surfaces of the opposite mucous membranes, as described earlier, the usual solitary occurrence and the sharp limitations, corresponding to the contacting areas of two spherical surfaces.

The clarification of the etiology permits the replacement of the provisionary name suggested by Heryng by a definite one and the naming of the ulcers on the grounds of their origin, "decubital or contact ulcers of the pharynx," as I suggested in 1932. Miyakogawa in his publication also accepted this new name for this particular condition of the pharynx.

SUMMARY

On the basis of a series of clinical observations an explanation for the development of the benign pharyngeal ulcer of Heryng is furnished, and accordingly, with regard to its genesis, this condition is given the name "decubital or contact ulcer of the pharynx."

De Merschman Gardens.

4. Miyakogawa, T.: Zwei Fälle von Pharynxgeschwür infolge Berührung, Oto-rhino-laryng **12**:209, 1939.

PRIMARY OSTEOMA OF THE FRONTAL SINUS

R. WALLACE TEED, M.D.
ANN ARBOR, MICH.

Osteoma originating in the confines of the frontal sinus is designated "primary" or "primitive," as opposed to the secondary type, which invades the sinus from without. An example of such a lesion is presented in the following case:

REPORT OF CASES

G. A., a white man aged 22, was admitted to St. Joseph's Mercy Hospital, Ann Arbor, Mich., June 8, 1938. He had been perfectly well until about two weeks before admission, when he caught cold. The infection was mild, and there were no unusual symptoms until six days before admission, when he began to note pain over the right eye. This continued for two or three days, after which he began to notice a fulness in the right upper lid. This gradually increased, so that when the patient was admitted to the hospital the lids were entirely closed. Pain had also increased and extended over the entire forehead and vertex. It had been practically constant and of a boring character. During the past week there had been a moderate amount of mucopurulent discharge from the nose, but no obstruction. There had been no complaints referable to the throat or ears. The patient was first seen by an oculist, Dr. A. S. Barr, who believed that the trouble was originating in the sinuses and referred him to me.

Physical Examination.—The patient appeared to be acutely ill. He was unable to open his right eye because of a red swelling of the lids, particularly the upper. The swelling also extended halfway up the forehead. The left eye appeared to be normal. No area of softening appeared on either the lid or forehead, and there was no discharge from the eye. Palpation revealed a tender, boggy swelling over the right supraorbital region, but no fluctuation could be detected. When the lid was opened, the conjunctiva was seen to be red and edematous, but no abscess could be made out. Tenderness was also present along the right side of the root of the nose. The nares were clean. The septum was slightly deflected to the left, but no obstruction was present. The turbinates were somewhat congested on both sides, particularly on the right. There was edema with a small amount of purulent discharge in the anterior portion of the right middle meatus. The left nasal fossa was quite clean, and the nasopharynx was normal. Transillumination of the antrums and the left frontal sinus gave negative results; swelling prevented examination of the right frontal sinus. The teeth showed several cavities. The tongue was heavily coated, the pillars of the fauces were injected and the tonsils were medium sized and septic. The larynx and ears were normal throughout. Examination of the heart, lungs and abdomen gave essentially negative results. The temperature was 101 F., the pulse rate 90 and respirations 22.

Read at a meeting of the staff of St. Joseph's Mercy Hospital, Ann Arbor, Mich., March 5, 1940.

Urinalysis gave negative results. Examination of the blood showed the erythrocyte count to be 4,460,000, the hemoglobin content 116 per cent and the leukocyte count 7,400, with lymphocytes 45 per cent, polymorphonuclears 53 per cent, eosinophils 1 per cent and monocytes 1 per cent. The Kahn reaction of the blood was negative. Culture of the nasal secretion revealed Streptococcus. Roentgen examination of the sinuses revealed a multilobulated opaque mass involving the right frontal and anterior ethmoid sinuses (fig. 1). The left frontal sinus and the maxillary, ethmoid and sphenoid sinuses appeared normal. In the lateral projection it appeared that the tumor either arose from the roof of the sinus or was in close contact with it.

A diagnosis of osteoma of the right frontal sinus with secondary infection and orbital cellulitis was made, and the patient was hospitalized. Conservative

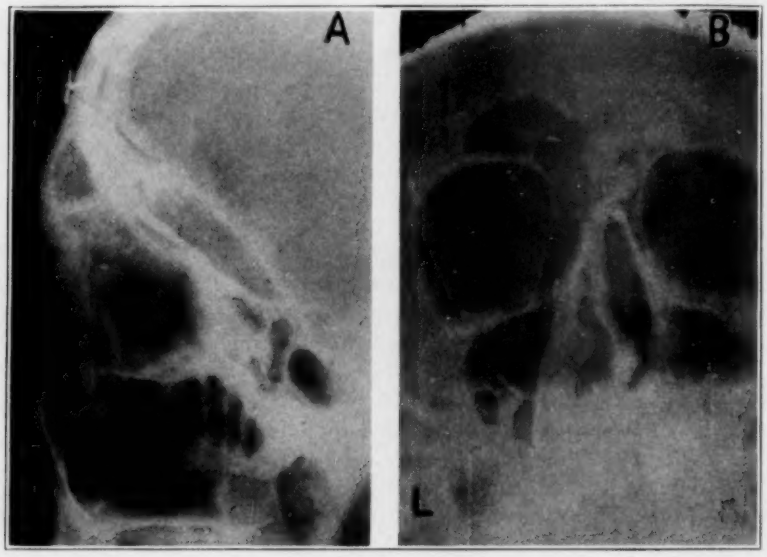

Fig. 1.—Roentgenograms of the patient before operation. *A*, lateral view. *B*, anterior-posterior view.

management, including continuous hot applications and the use of sedatives and fluids, was prescribed. The following day the patient felt well, with little pain and a normal temperature. The local application of heat was continued. Two days later (June 10) the orbital swelling had subsided considerably, and the pain had entirely disappeared. Improvement continued uninterruptedly, and the patient was discharged from the hospital on June 14. At this time there was no fever, pain or swelling, and the patient was instructed to engage in no strenuous activity while at home. He was seen at intervals in the office and on July 6 was readmitted for removal of the tumor. At this time there was no evidence of infection externally, but the right upper eyelid appeared to be drooping a bit, and on palpation a small firm elastic mass about 0.5 cm. in diameter could be felt in the upper inner angle of the orbit. Since the roentgenogram did not reveal the osteoma in this region, it was felt that this was probably a mucocele or a pyomucocele.

Since a culture of Streptococcus had been reported, the patient was put to bed and saturated with sulfanilamide for two days. Operation was carried out on July 8, with the use of local anesthesia. The right sphenopalatine ganglion and internal nasal nerve were cocainized, and 2 per cent procaine hydrochloride was injected along the right brow and the right side of the nose. A similar injection was made into the medial and superior portion of the orbit. An incision was then made from the outer third of the orbit, along the brow and down the lateral wall of the nose, to approximately the end of the nasal bone. The periosteum over the roof of the orbit was then elevated, in which procedure a small bulging mass was noted near the internal angle. This was filled with a purulent mucoid secretion. The frontal sinus was then entered with a gouge, and immediately a white hard bony tumor came into view. There was very little space between the tumor and the wall of the sinus, but two layers of membrane could be seen very clearly. Pus was also present in the sinus. The floor of the sinus was removed in its entirety, in the hope that the tumor could be extirpated without disturbing the anterior wall, but this was found to be impossible, as the tumor was firmly lodged within the sinus. Consequently, about 1.5 cm. of the anterior wall was removed. At this time the tumor was grasped with a rongeur and rocked free from its point of attachment, which was the medial septum between the two frontal sinuses. Most of the septum came away with the tumor, and the remainder was bitten away with the rongeur. The mucosa of the right frontal sinus was much thickened and was removed in its entirety. The membrane in the left sinus was also thickened and infected, but it was not removed. No exposure of the dura was made. A few small anterior ethmoid cells were then removed, and a large rubber tube was passed from the nose into the right frontal sinus. A Penrose drain was placed in the lower angle of the wound, which was closed in layers. The patient returned to bed in good condition.

The Penrose drain was removed in twenty-four hours, and all sutures in forty-eight hours. The postoperative swelling of the eyelid subsided rapidly, and the wound healed by first intention. The patient was allowed to be up on July 16 and was discharged on July 22. The large rubber tube was allowed to remain in the frontal sinus for three weeks, after which time it was removed. The patient has been seen on numerous occasions during the past eighteen months, and although he has had several infections of the upper respiratory tract, no evidence of trouble in the right frontal sinus has developed. No evidence of recurrence has appeared (fig. 2).

A culture of material from the frontal sinus at operation revealed staphylococci with a few diphtheroids. A gross diagnosis of eburnated osteoma was made by the hospital pathologist, Dr. S. C. Howard, but unfortunately the specimen was lost during preparation for sectioning.

An additional patient was admitted to the service of Dr. A. C. Furstenberg, who has given me permission to include a report of the case here.

The patient, B. C., a white man aged 20, was admitted to St. Joseph's Mercy Hospital on July 29, 1929. He stated that when he was a small child he was struck over the left eye with a sledge hammer. A few years later he was injured in the same place when he fell off a bicycle. No symptoms appeared, however, until about seven years before admission, when a tumor was noted over the left brow. This grew slowly at first, but in the last year the growth had been very

rapid. There had been no headaches or visual disturbances and no symptoms relative to the nose or ears. The general condition had been good.

Examination revealed a firm, painless swelling over the left eye, which extended from the midline laterally to the temple and upward over the supraorbital ridge for about 1½ inches (4 cm.). It protruded beyond the surrounding frontal bone about ¾ inch (2 cm.) and was about the size and shape of a medium-sized oyster shell. The examination of the nose, throat, ears, chest and abdomen gave negative results. Roentgen examination revealed a large bony tumor molding the left frontal sinus, which was considerably enlarged.

A diagnosis of osteoma of the left frontal sinus was made, and the patient was admitted to the hospital for operation, which was carried out by Dr. Furstenberg on July 30. With the patient under anesthesia obtained by the use of ether,

Fig. 2.—Postoperative photographs of the patient. *A*, front view. *B*, close-up view showing the postoperative result.

an incision was made in the left brow, and a second, at right angles, was carried upward in the midline. The tumor was exposed under the periosteum. A portion of the supraorbital ridge was excised, and the tumor was elevated with a gouge. The dura was exposed but not torn. There was considerable bleeding, which was controlled by packing. The incision was closed with dermal sutures, and a small drain was placed in the bottom of the wound. The drain was removed in two days, and all sutures on August 5. The temperature remained normal throughout, and healing occurred by first intention. The patient was discharged on August 9. No pathologic examination of the tumor was made.

Bony growths of various kinds have been seen and described for hundreds of years, and while all of the bones of the body have been

attacked, it is generally conceded that the bones of the skull are the most frequent site of the lesion. Some of the famous "horned men" of history have been victims of such bony growth, and the term "spina ventosa" or "wind-horn" is of great antiquity.

Bony tumors growing within the cranial cavity, however, have been recognized only during the past three hundred years. Bartholinus, who probably saw the first case, according to Hewett (1867), was said to believe that he was dealing with a "petrified brain," because of the lobulations on its surface. Du Verney (also quoted by Hewett, 1867), a few years later, actually described in a similar case the lobes, hemispheres, convolutions, pineal gland and vermiform process. He even imagined that he saw the gray and white matter, completely turned to stone. It remained for Vallisnieri, in 1733, to demonstrate that these lesions were not petrified brains, but simply bone tumors.

According to Velpeau (1847), Bartholinus, Fabricius Hildanus and Schenckius noted "calculi" in the frontal sinuses in cadavers, but the first observation of a tumor of the frontal sinus during life was made by a Veiga in 1506, although it was not published until 1586. The patient, a woman, presented a tumor as large as a hen's egg in the superointernal portion of the orbit, which caused exophthalmos and considerable facial deformity. a Veiga removed the tumor in pieces, and healing resulted. The patient's vision and facial appearance returned to normal.

Vallisnieri (1733) described a skull in the museum at Florence, Italy, in which an osteoma of the frontal sinus had extended into the frontal lobes of the brain. It had become detached from the walls of the sinus by necrosis.

In 1736, Petit described an orbital tumor in the following words:

"J'en ai vu une au bord superieur de l'orbite, du cote du petit angle de l'oeil, laquel avoit eleve la paupiere et faisoit sur le globe de l'oeil une si grand saillie, que les cils de la paupiere etoient sur le sommet apparent de la tumeur, et la partageoient en deux, de maniere que la moitie superieure etoit couverte par la peau de la paupiere, et l'inferieure appuyant le globe de l'oeil etoit couverte par le conjonctive. Le maladie devenu louche voyait les objets doubles." (I have seen [such a tumor] on the superior border of the orbit near the small angle of the eye, which had elevated the lid and caused the globe of the eye to protrude so much that the eyelashes were on the apparent summit of the tumor and divided it into two parts, the upper half being covered by the lid, and the lower half, resting on the globe, being covered by conjunctiva. The patient, because of the squint, developed diplopia.)

Since operation or autopsy was not mentioned, it is impossible to be definite as to the origin of the tumor, and it is not included in the present series. The clinical description, however, is excellent.

An interesting case was reported by Professor Sporing in Albrecht Haller's book in 1758, although it has been credited by others to Bell,

Acrel and Middlemore, all of whom described it in their books. Sporing wrote:

"Among my papers I find an account of a very extraordinary osseous excrescence near the eye of the peasant, which after torturing the poor man almost fourteen years, fell out spontaneously. . . . Olof Laitin, a peasant of Ahala . . . was in the thirty-fifth year of his age seized with violent pains in his head, accompanied by an inexpressible heat, Shootings, and Irritations, chiefly over his eyebrows; at the end of a month there suddenly appeared betwixt the Nose and the right Eye, a hard Nodus of the Bigness of a small Bean, with an aqueous Liquor copiously issuing from that Eye. This Osseous Excrescence, which afterwards grew . . . remained from Novem. 1724, to March 1738. During its growth the Eye-ball was insensibly forced from its natural Position, that at length it came to project out at the opposite Corner of the Eye, and was a Disfigurement not to be viewed without Disgust; the Tubercle, in the meantime, was surrounded with a pus continually dripping at his right Nostril. As the Excrescence enlarged the sight of the Eye grew weaker, till it was very near a total extinction. A Camp-Surgeon in the Neighborhood was for taking out the bone by Exfoliation, of which some vestiges are to be seen in the Bone . . . but the Wound beginning to bleed extremely, and the Patient fainting under the Severity of the Operation it was judged advisable to desist. In December 1836, Olof Ikain a Peasant of Pixema an adjacent Parish, and noted for his Cures, offerred to try his skill for the Relief of the poor man, beginning with an incision around the Bone which also caused a great effusion of Blood, and afterwards applying to it something carefully concealed, but, the Day following, so violent in its Operations that without help the Patient could not go from one Room to another: After twelve days of exceeding Pain, attended with Deliquiums, he grew tolerably well, and some Months after in the Spring, 1737, had the courage to venture a second Time on the erosive, but with the like excruciating Consequences. In March, 1738, this Osseous Congeries drop off of itself, the Pain immediately ceased; the Eye mends daily, and is gradually returning to its socket. The Aperature made by the Discharge of the Bone, though still suppurating, is in a fair Way of soon being healed."

Brassant (1774) attacked the tumor in his case with caustic. After three or four months of suppuration, the tumor exfoliated, along with much of the tumefied bone around it. The eye returned to its normal position, and healing was complete some time later.

The case reported by van der Meer in 1829 is a monument not only to the surgeon but to the courage and fortitude of his patient as well. The latter, a man of 54, had a large bony tumor of many years' standing, which occupied the whole of the upper and inner part of the right orbit. The eye was displaced downward and outward on the cheek, and vision was entirely destroyed. van der Meer extracted the globe and two days later exposed the tumor. Repeated attacks with trephine, hammer and chisels were made, but very little impression could be made on the stony-hard mass. An attempt to soften the tumor with acid having failed, the surgeon operated daily for eight weeks, removing each day a little of the tumor with chisels. The greater part of the orbital

portion was thus removed, with the result of considerable improvement in the appearance of the patient.

The case reported by Hilton (1836) is of interest because of the great size of the tumor (442 Gm.), because of its spontaneous detachment without surgical intervention and because it is the first case in which a picture of the patient was published (fig. 3).

The first case in which surgical intervention was employed in this country was that which Knapp reported in 1880. He had reported another case in 1861, but the patient was seen and the operation was performed in Heidelberg.

Fig. 3.—A reproduction of the first illustration showing a patient who has had osteoma (Hilton's case, 1836).

Birch-Hirschfeld (1930) defined an exostosis as a circumscribed neoformation of bone which leads to the development of rounded or pointed outgrowths. A generalized or diffuse hyperplasia of bone was spoken of as a hyperostosis. If the neoplasm occurs within a bony cavity, he said he believed it should be referred to as an enostosis. To the latter class belong the osteomas. Most of the reports prior to 1890 refer to these growths as exostoses, although the term "osteoma" had been proposed by Hooper in 1828. Since 1890, however, the name "osteoma" has had practically universal usage.

Considerable debate has centered around the relative frequency of osteoma of the frontal sinuses. Before the advent of the roentgen ray,

it was considered extremely rare, but in recent years some authors have expressed the opinion that it was fairly common. Andrews (1887) reviewed 429,989 cases of ocular disease at three institutions, the New York Eye and Ear Infirmary, the New York Ophthalmological and Aural Institute and the Manhattan Eye, Ear, and Throat Hospital and found only 8 orbital exostoses, of which 2 or 3 probably originated within the frontal sinuses. Faulkner (1924) stated that in twenty years at the last-named institution he had seen but 3 cases of osteoma of the frontal sinus. Between 1918 and 1939 only 2 cases were seen among 48,000 patients admitted to the general services of St. Joseph's Mercy Hospital, Ann Arbor. Moreover, many otolaryngologists have spent a lifetime in practice without seeing a case. The great majority of reports have been of single cases, there being only twenty reports with 2 (Home, 1799; Lambl, 1857; Arnold, 1873; Hutchinson, 1888; Kikusi, 1888; Mitvalsky, 1894; Hamilton, 1901; Manasse, 1910; Boardman and Donovan, 1918; Harper, 1927; Fröding, 1929; Sackin, 1929; Birch-Hirschfeld, 1930; Brown, 1930; Armitage, 1931; Carmody, 1932; Roger, 1932; Neumann, 1934; Matolcsy, 1936, and di Marzio, 1936), six with 3 cases (MacKenzie, 1854; Mackenty, 1924; Claus, 1927; Harris, 1928; Fetissof, 1929, and Causse, 1934), one with 4 cases (Carmody, 1935), one with 6 (Coppez, 1895), one with 9 cases (Childrey, 1939) and one with 30 cases (Sättler, 1938).

The total number of cases, likewise, has been a matter of considerable difference of opinion. Bornhaupt (1881) collected 23 cases, while Tauber (1898), seventeen years later, listed only 20. Lagrange, in 1904, found 121 cases, while Hucklenbroich, in 1905, included only 55. Gerber, in 1907, listed only 84 cases, and Boenninghaus, in 1914, 75. Dahmann (1922) listed 88 cases; Rhigetti (1926), 95 cases; Harris (1928), 117 cases; Eckert-Möbius (1929), 113 cases; Fetissof (1929), 105 cases; Birch-Hirschfeld (1930), 120 cases; Armitage (1931), 119 cases; Roger (1932), 140 cases; di Core (1933), 160 cases; Causse (1934), 134 cases; Carmody (1935), 103 cases; Gatewood and Settel (1935), 147 cases, and Malan (1938), 178 cases. Many of the authors cited apparently accepted the figure of a previous author and added to that the number of cases found in the indexes in the interim. In view of the resulting confusion, Carmody (1935) stated: "Any accurate estimate of the exact number of cases would require a careful check of the reports and tabulation of cases. Such a careful survey might be of real value."

I have undertaken such a survey and have been able to collect 321 cases (table 1). There are, in addition, possibly a dozen other reports in the literature which were unavailable to me. There were reports of 2 cases in the Polish literature, 3 in the Russian and 1 each in the Japanese, Hungarian, Italian, Swedish, German, French and Czechoslovakian. A few of the cases prior to 1851 are somewhat doubtful, but a careful

reading of the reports has led to the conclusion that there are sufficient grounds for including them here.

Consideration of the data indicates that osteoma of the frontal sinus is certainly not a rare lesion but that, on the other hand, it cannot be spoken of as common.

The frontal sinus is the most frequent site for the development of osteoma of the nose and the paranasal sinuses. Malan, who has written the best of the recent reviews, collected 458 cases: in 178 (38.95 per cent) the growth occurred in the frontal sinuses; in 110 (23.85 per cent), in the ethmoid sinuses; in 41 (8.97 per cent) in the maxillary sinuses; in 22 (4.81 per cent), in the nasal cavity, and in 7 (1.53 per cent), in the sphenoid sinuses. In 41 (8.97 per cent) the exostosis was external; in 32 (7 per cent) the origin was doubtful, and in 27 (5.9 per cent) the first cavity involved was uncertain.

Practically all authors have commented on the fact that osteoma of the frontal sinus is essentially a disease of youth. Childrey (1939), however, in a group of 9 patients found the average age to be 46.4 years. The youngest patient in the present series was 13 (Carmody, 1935), while the oldest was 75 (Patriarca and Rossello, 1934). Table 2 gives the number of cases in each decade:

These figures agree well with those given by Eckert-Möbius (1929).

The age of the patient when consultation is sought gives little clue to the time at which the lesion began, except that in general it is usually during the second and third decenniums. In several cases, however, no signs appeared until after 50. The development of the tumor may be slow or rapid; it may grow for a time and then remain dormant over a period of years, only to resume its growth for no apparent reason. In table 1 I noted the age at which symptoms or signs appeared, but nothing significant can be gained from a study of these figures.

Most authors have agreed that males are more susceptible to osteoma than females. Eckert-Möbius (1929) found 110 cases in males and 70 in females. In the present series, the proportion was 172 to 93.

It might be of interest to review the various theories as to the causation of osteoma in the light of the experience accumulated to date.

The original idea of Bartholinus and Du Verney relative to petrified brains is, of course, too fantastic to warrant further discussion. The further idea that calculi formed in the frontal sinuses in a manner similar to that of their formation in the urinary bladder also can receive no serious attention.

Home (1799), after studying the teeth of elephants and other large animals, concluded that exostoses of the sinuses were formed on vascular excrescences of the mucosa in a manner similar to that of the development of teeth on their pulp. Further studies on teeth have elucidated this process further, so that no possible parallel can be drawn.

TABLE 1.—*Incidence of Osteoma of the Frontal Sinus*

Date	Author	Sex of Patient	Age	Age at Onset	Trauma	Operation	Attachment	Comment
1586	a Veiga.....	F	..	..	....	Removal	.....	Recovery
1733	Vallisnieri....	..	..	..	....	None	.....	Pathologic specimen
1758	Sporing.....	M	49	35	No	Operation stopped; later, treatment with caustic	.....	Exfoliation; recovery
1774	Brassant.....	F	30	15	....	Treatment with caustic	Frontoethmoid region	Exfoliation; recovery
1799	Home.....	..	..	..	....	None	.....	Pathologic specimen
	Idem.....	..	..	..	....	None	.....	Pathologic specimen
1805	Lucas.....	F	28	..	Yes	Unsuccessful; removal following suppuration	.....	Recovery
1812	Baillie (specimen 795 in Hunterian Museum [Royal College of Surgeons of England])	..	..	..	....	None	.....	Pathologic specimen
1829	van der Meer	M	54	..	....	Operation daily for 8 weeks	.....	Recovery
1830	Salzer.....	F	17	13	Yes	Unsuccessful; exostosis removed later	.....	Recovery
1832	Oesterlen.....	M	59	31	Yes	None	.....	Died at 76 of pneumonia
1834	Amussat.....	F	52	42	No	Recurrence after operation	.....	Died 1 year later of brain abscess
1836	Hilton.....	M	36	13	No	Spontaneous exfoliation	.....	Recovery
1841	Bouyer.....	M	28	24	Yes	Removal	Floor	Recovery
1844	Rokitansky..	..	26	..	....	None	.....	Pathologic specimen
1845	Adelmann....	F	23	..	No	Removal followed by meningitis	.....	Recovery
1851	Canton.....	F	25	..	No	Removal	.....	Recovery
1851	Ilmoni and Törnroth	F	50	..	No	Partial removal; rest exfoliated 3 mo. later	.....	Recovery
1851	Weiss (Roux's case)	F	Young	..	....	Unsuccessful	.....	Death
1853	Walton.....	M	40	Boy	Yes	Removal	.....	Recovery
1854	Busch.....	F	22	15	No	Removal	.....	Patient died 13 days later of brain abscess
1854	Mackenzie (p. 48, case 68)	M	..	..	....	Removal in 2 operations	Anterior wall	Recovery
1854	Idem (p. 79)	..	..	..	....	None	.....	Pathologic specimen
1854	Idem, ibid....	..	..	..	....	None	.....	Pathologic specimen
1856	Lenoir.....	F	28	25	No	Removal	Frontoethmoid region	Recovery
1857	Lambl (Vrolik's case)	M	36	14	....	None	.....	Pathologic specimen
	Idem (Pitha's case)	M	21	..	....	None	.....	Pathologic specimen; patient died of "lung edema"

TABLE 1.—*Incidence of Osteoma of the Frontal Sinus—Continued*

Date	Author	Sex of Patient	Age	Age at Onset	Trauma	Operation	Attachment	Comment
1860	Demarquay..	M	34	30	Yes	Removal	Anterior wall	Recovery
1860	von Oettingen	M	24	21	....	Partial removal	.....	Patient died 29 days later of brain abscess
1861	Knapp.....	M	14	..	No	Partial removal	.....	Patient died of meningitis
1864	Björkén.....	M	21	18	....	Partial removal	.....	Patient died on 9th day of meningitis
1864	Warren Museum (cited by Armitage, 1931)	M	Young	3 yr. before admission to hospital	....	Unsuccessful	.....	Patient died of meningitis and brain abscess
1864	Virchow	M	47	44	No	None	Floor	Pathologic specimen
1865	Dolbeau	M	21	18	No	Removal	Septum	Recovery
1865	Textor	F	34	32	....	Removal	.....	Recovery
1867	Hewett	F	20	..	....	Partial removal	.....	Recovery
1867	Lawrence	..	..	..	....	None	.....	Pathologic specimen
1870	Paget (specimen in museum, Cambridge University)	..	..	..	....	None	.....	Pathologic specimen larger than skull
	Idem (case of Turner, 546-a in anatomic museum University of Edinburgh)	..	..	..	....	None	Floor	Pathologic specimen
1871	Birkett	F	15	13	No	Partial removal	Posterior wall	Patient died of brain abscess
1871	Jobert.....	..	..	..	....	Unsuccessful	Anterior wall	Death
1871	Letenneur....	F	40	..	....	Removal	.....	Recovery
1871	Richet.....	M	20	20	....	Removal	.....	Recovery
1871	Verneuil.....	M	23	20	No	Partial removal	Posterior wall	Recovery
1872	Bryant.....	M	24	19	No	Removal	Anterior wall	Recovery
1873	Arnold.....	M	23	..	....	None	Posterior wall	Death
	Idem (operation by Professor Simon)	M	26	20	No	Partial removal	.....	Patient died in 2 weeks of meningitis and brain abscess
1874	Banga (operation by Professor Socin)	M	18	15	Yes	Removal	Floor	Patient died in 10 days of meningitis and brain abscess
1875	Burow	F	58	28	No	Removal after 4 yr. of suppuration	.....	Recovery
1878	de Vincentiis	M	35	..	....	None	Superior wall	Pathologic specimen
1880	Berlin.....	F	..	..	....	Enucleation of the eye	.....	.....
1880	Knapp.....	M	48	..	No	Removal	Septum	Recovery
1881	Bornhaupt (operation by Professor Volkmann)	F	18	16	No	Removal	.....	Recovery
1881	Solger.....	..	62	..	....	None	.....	Pathologic specimen
1882	Imre.....	F	64	22	....	Treatment with caustic; later spontaneous exfoliation	.....	Recovery

TABLE 1.—*Incidence of Osteoma of the Frontal Sinus—Continued*

Date	Author	Sex of Patient	Age	Age at Onset	Trauma	Operation	Attachment	Comment
1882	Teillais.....	M	19	..	Yes	Partial removal	.....	Recovery
1882	Tweedy.....	M	25	23	No	Partial removal	.....	Patient died in 1 mo. of meningitis
1883	Panas.....	F	18	13	No	Partial removal	Floor	Patient died in 6 days of meningitis
1883	Weinlechner..	M	51	25	No	Removal	.....	Recovered; patient later died of erysipelas after removal of nasal osteoma
1885	Panas.....	F	49	4	No	None	.....	.....
1885	Tillmanns....	F	53	35	No	Removal	Fronto-ethmoid region	Recovery
1886	Lucas-Championnière	M	..	..	No	Removal	.....	Recovery
1887	Andrews.....	F	19	18	No	Removal	Septum	Recovery
1887	Grossman....	M	24	..	....	Partial removal	.....	Recovery
1888	Hutchinson..	F	Young	..	....	Partial removal; rest enucleated after suppuration	.....	Recovery
	Idem.....	M	Young	..	....	Removal	.....	.....
1888	Kikusi (operation by Professor Bruns)	M	26	20	....	Removal	.....	Recovery
	Idem.....	M	45	19	....	Removal	.....	Recovery
1888	Reeve.....	..	..	..	....	Removal	.....	Recovery
1888	Silcock.....	F	20	..	No	Partial removal	.....	Recovery
1888	Yates.....	..	..	..	....	None	.....	Pathologic specimen
1889	Berg.....	M	37	..	....	Removal of 13 tumors	.....	Recovery
1889	Bessel-Hagen	F	26	23	....	Removal	Fronto-ethmoid region	Recovery
1889	Jamain.....	F	16	..	....	Removal	.....	Recovery
1890	Adamük.....	M	22	..	....	Removal	.....	Recovery
1890	Boyd.....	M	20	13	....	Removal	Posterior wall	Recovery
1890	Ischunin.....	..	..	..	....	Removal	.....	.....
1892	Maydl.....	F	15	12	Yes	Removal	.....	Recovery
1892	Nakel.....	F	18	15	....	Removal	Fronto-ethmoid region	Recovery following erysipelas
1892	Poppert (operation by Professor Bose)	M	26	17	....	Removal	Posterior wall	Recovery
1892	Prochnov....	M	19	16	....	Partial removal	.....	Recovery
1894	Mitvalsky.... (case 2)	M	30	26	Yes	Partial removal	.....	Recovery
1895	Coppez.....	M	20	18	....	Partial removal	.....	Recovery
	Idem.....	F	65	63	....	Removal	.....	Recovery
	Idem.....	M	25	19	....	Removal	.....	Recovery
	Idem.....	M	22	19	Yes	Removal	.....	Recovery
	Idem.....	F	20	18	....	Removal	Anterior wall	Recovery
	Idem.....	M	48	46	....	Refused	.....	.....
1895	Martin (operation by Dudon)	M	23	..	....	Removal of detached tumor	None	Recovery
1896	Coppez.....	M	19	19	....	Removal of mucocele only	.....	Recovery

TABLE 1.—*Incidence of Osteoma of the Frontal Sinus—Continued*

Date	Author	Sex of Patient	Age	Age at Onset	Trauma	Operation	Attachment	Comment
1896	Sattler.....	M	25	22	....	Removal	.....	Healed; 8 mo. later another tumor removed
1896	Thomas.....	F	46	33	Yes	Removal	Roof	Recovery
1898	Fridenberg...	..	..	..	....	Removal	Posterior wall	Recovery
1898	von Navratil	M	26	23	....	Drainage only	.....	Recovery
1898	Tauber.....	M	33	16	Yes	Removal of 3 tumors	1 to posterior wall; 2 detached	Recovery
1899	Coppez.....	F	20	18	Yes	Removal	.....	Recovery
1899	Luc.....	F	56	..	....	Removal	Fronto-ethmoid region	Recovery
1899	Moser.....	F	22	20	Yes	Removal	Septum	Recovery
1899	de Santi.....	..	..	..	....	None	.....	Tumor movable
1899	Strakhoff....	M	30	..	....	Removal by patient	.....	Recovery
1900	Pergens.....	..	..	..	....	Removal	.....	Recovery followed by buphthalmia
1900	Witzheller....	M	28	27	No	Partial removal	Posterior wall	Recovery, complicated by cavernous sinus thrombosis
1900	Zimmerman..	M	17	16	....	Removal	Fronto-ethmoid region	Recovery
1901	Hamilton....	M	36	14	Yes	Removal	.....	Recovery
	Idem.....	M	27	..	....	Removal	.....	Recovery
1902	Giese.....	F	35	34	....	Removal	.....	Recovery
1902	Kan.....	..	..	..	....	Removal	.....	Recovery
1903	Ferenczi.....	..	..	..	....	Removal	.....	Recovery
1903	Manninger...	..	..	..	....	Removal	Posterior wall	Recovery
1903	Rollet.....	F	68	..	....	Removal	.....	Recovery
1903	Sipocz.....	..	..	..	Yes	Removal	.....	Recovery
1904	Axenfeld.....	M	40	35	....	Removal	Septum	Recovery
1904	Birch-Hirschfeld (operation by Perthes)	F	26	22	....	Removal	Fronto-ethmoid region	Recovery
1904	Goyanes.....	F	24	19	....	Removal	Posterior wall	Recovery
1904	Haenel.....	M	40	31	....	Removal	Posterior wall	Recovery; tumors also removed from antrums
1905	Hastings.....	..	..	..	....	None	Floor	Pathologic specimen
1905	Taylor.....	M	16	..	....	Removal	.....	Patient died in 1 week of meningitis
1906	Jones and Holland	F	35	20	....	Removal	Postero-superior wall	Recovery
1906	Oppenheimer	F	42	..	....	Drainage only	.....	Recovery
1907	Davies.....	M	..	..	Yes	Removal	Postero-inferior wall	Recovery
1907	Gerber.....	M	24	..	....	Removal	.....	Recovery
1907	Kirchoff.....	M	31	..	....	Removal	.....	Recovery
1907	Rick (operation by Küster)	M	18	18	....	Removal	Floor	Recovery
1908	Marx.....	F	28	18	....	Removal	Floor	Recovery
1908	Milligan.....	M	70	69	....	Removal	Floor	Recovery

TABLE 1.—*Incidence of Osteoma of the Frontal Sinus—Continued*

Date	Author	Sex of Patient	Age	Age at Onset	Trauma	Operation	Attachment	Comment
1908	Scimemi.....	..	..	..	....	Removal	.....	Recovery
1909	Ardenne.....	M	64	40	No	Removal	Fronto-ethmoid region	Recovery
1910	Manasse.....	M	16	..	....	Removal	Posterior wall	Recovery
	Idem.....	..	..	..	....	None	.....	Pathologic specimen
1911	Dubar and Potel	M	..	..	....	Removal	Posterior wall	Recovery
1911	Van Wagenen	F	49	40	....	Partial removal	.....	Recovery
1912	Cargill.....	M	22	..	Yes	None	.....	Diagnosis by roentgen examination
1912	Handley.....	M	35	32	....	Removal	Posterior wall	Recovery
1912	Pfeiffer.....	M	24	..	No	Removal	Floor	Recovery
1912	Spasski.....	F	18	..	....	Removal	.....	Recovery
1913	Citelli.....	M	15	14	....	Removal	Floor	Recovery
1913	Schlesinger...	F	29	..	....	None	.....	Patient died of glioma
1914	Gerber.....	..	..	..	....	.....	.....	.....
1914	Golovin.....	M	..	..	....	Removal of 3 tumors	.....	.....
1914	Horowitz....	..	..	..	....	Removal	.....	Recovery
1914	Weingärtner	M	42	..	....	None	.....	Diagnosis by roentgen examination
1914	Zumhasch...	M	23	8	Yes	Removal	Floor	Recovery
1915	Lack.....	..	..	..	....	Removal	Posterior wall	Recovery
1915	Spicer.....	..	..	..	....	Removal	.....	Recovery; recurrence
1915	Wray.....	..	..	..	....	None	.....	Pathologic specimen
1916	Chapman....	F	52	49	....	Removal	Anterior wall	Recovery
1916	Probert.....	M	15	..	....	Removal	Anterior wall	Recovery
1916	Veasy.....	F	30	19	....	None	.....	Diagnosis by roentgen examination
1917	Jackson.....	..	..	..	....	.....	.....	.....
1917	Reeve.....	F	23	22	....	Removal	.....	Recovery
1918	Barnhill.....	F	16	..	....	Removal	Fronto-ethmoid region	Recovery
1918	Boardman and Donovan	M	..	..	....	Removal	.....	Recovery
	Idem.....	M	Young	..	....	Removal	.....	Patient died of meningitis
1918	Culbert.....	M	43	..	....	Partial removal	.....	Patient died of brain abscess
1918	Hofmeister..	M	47	..	....	Removal	.....	Recovery
1918	Mackenty....	M	..	..	....	None	.....	Patient died of meningitis
1918	Sewall.....	M	25	..	....	Removal	Anterior wall	Recovery
1918	Thomas.....	M	14	14	Yes	Removal	.....	Recurrence
1918	Tod.....	M	23	..	....	Partial removal	.....	Recurrence
1919	Jacques.....	M	21	20	....	Removal	Septum	Recovery
1919	Lang and Armour	M	19	19	....	Removal	.....	Recovery
1919	Lange.....	F	18	18	....	Removal	Posterior wall	Recovery
1919	Stolpe.....	M	21	21	....	Removal	Anterior wall	Recovery

TABLE 1.—*Incidence of Osteoma of the Frontal Sinus—Continued*

Date	Author	Sex of Patient	Age	Age at Onset	Trauma	Operation	Attachment	Comment
1922	Claridge.....	..	..	..	....	None	.....	Pathologic specimen
1922	Dahmann....	F	27	..	....	Removal	Fronto-ethmoid region	Recovery
1922	Erb.....	..	..	..	....	Unsuccessful	.....	Recovery
1922	Latteri.....	M	26	..	....	Removal	.....	Recovery
1922	Lindenmeyer	F	..	..	....	Removal	.....	Recovery
1923	Reh.....	F	16	14	....	Removal	.....	Recovery
1924	Babcock.....	M	30	28	Yes	Removal	Septum	Recovery
1924	Faulkner.....	M	..	..	Yes	Removal	Posterior wall	Recovery
1924	Howarth.....	M	38	..	....	Removal	Fronto-ethmoid region	Recovery
1924	Mackenty....	..	..	..	....	.....	.....	.....
	Idem.....	..	..	..	....	.....	.....	.....
	Idem.....	..	..	..	...	.....	.....	.....
1924	Nikolskij.....	..	..	..	....	Removal in 2 operations	.....	Recovery
1924	Piazza.....	F	22	22	....	Unsuccesful	.....	Recovery
1924	Versteegh....	F	14	..	....	Removal	Posterior wall	.....
1924	Vulowitsch...	M	20	18	Yes	Removal	Septum	Recovery
1924	Watson.....	M	29	15	....	Removal	.....	Recovery
1925	Bacher.....	M	25	..	Yes	Removal	Junction of anterior and superior walls	Recovery
1925	Buys.....	F	23	23	....	Removal	.....	Recovery
1925	Goodyear....	M	32	30	....	Removal	Supero-lateral wall	Recovery
1925	Lee.....	M	17	16	Yes	Partial removal	.....	Recovery
1925	Schousboe...	F	54	..	....	Removal	.....	.....
1926	Mouret and Dejean	M	36	28	Yes	Removal	.....	Recovery
1926	Lebensohn...	M	24	20	....	Removal	.....	Recovery
1926	Lillie and Anderson	..	..	..	....	Removal	Fronto-ethmoid region	Recovery
1926	Rhigetti.....	M	16	15	....	Removal	Fronto-ethmoid region	Recovery
1926	Serra.....	F	56	..	No	Removal	.....	Recovery
1927	Caccialupi...	F	23	..	....	Removal	Detached	Recovery
1927	Claus.....	F	48	..	....	.....	.....	Diagnosis by roentgen examination
	Idem.....	M	51	..	....	.....	Anterior wall	Diagnosis by roentgen examination
	Idem.....	..	..	..	....	None	Posterior wall	Pathologic specimen
1927	Garretson....	F	19	..	....	Removal	.....	Recovery
1927	Harper.....	F	17	16	Yes	Removal	Posterior wall	Recovery
	Idem.....	M	44	44	....	Removal	.....	Recovery
1927	Mouret and Dejean	M	38	..	....	Removal	Septum	Recovery
1927	Neilson.....	M	43	..	....	Refused	.....	.....
1927	Neumann....	F	Old	..	....	Removal	Fronto-ethmoid region	Patient died of brain abscess
1927	Riecke.....	M	19	19	....	Removal	Fronto-ethmoid region	Recovery
1927	Suchanek....	M	59	..	....	Partial removal	.....	Recovery
1928	Acker.....	M	27	..	....	Removal	None seen	Recovery

TABLE 1.—*Incidence of Osteoma of the Frontal Sinus—Continued*

Date	Author	Sex of Patient	Age	Age at Onset	Trauma	Operation	Attachment	Comment
1928	Claus.....	..	..	..	....	Removal	Fronto-ethmoid region	Recovery
1928	Harris.....	M	30	27	....	Removal in 2 operations	.....	Recovery
	Idem.....	M	..	..	....	Removal	.....	Recovery.
	Idem.....	M	30	..	....	Refused	.....	.....
1928	Rodger.....	..	..	..	....	Removal	.....	Recovery
1929	Browder.....	M	16	13	....	Removal	Fronto-ethmoid region	Recovery
1929	Fetissof.....	M	24	16	....	Removal	.....	Recovery
	Idem.....	M	20	12	....	Removal	.....	Recovery
	Idem.....	F	19	16	....	Removal in 2 operations	.....	Recovery
1929	Fröding.....	..	..	..	....	None	.....	Diagnosis by roentgen examination
	Idem.....	..	..	..	....	.....	.....	.....
1929	Howarth.....	F	29	..	....	Partial removal	.....	Recovery
1929	Kupermann..	..	..	..	....	.....	.....	.....
1929	Nylen.....	M	..	..	Yes	None	.....	Diagnosis by roentgen examination
1929	Rosenstein...	F	36	35	....	Removal	.....	Recovery
1929	Sackin.....	..	..	..	....	None	.....	Diagnosis by roentgen examination
	Idem.....	M	35	..	....	None	.....	Diagnosis by roentgen examination
1929	Simeoni.....	F	15	..	....	Removal	Floor	Recovery
1929	Tyczka and Chorobski	F	62	..	....	.....	.....	.....
1929	Vitte.....	F	26	..	Yes	Removal	Anterior wall	Recovery
1929	Zeliger.....	M	37	13	....	Removal	.....	Recovery
1930	Birch-Hirschfeld	F	26	..	....	Removal	.....	Recovery
	Idem.....	F	18	..	....	Removal	Posterior wall	Recovery
1930	Beck.....	M	49	..	....	Removal	.....	Recovery
1930	Brown.....	M	34	34	....	Removal	Floor	Recovery
	Idem.....	..	..	..	....	None	.....	Diagnosis by roentgen examination
1930	Eckert-Möbius	M	22	..	....	Removal	.....	Recovery
1930	Leavenworth	M	23	..	....	Drainage only	.....	Recovery
1930	Neumann....	M	20	20	....	Removal	Lateral wall	Recovery
1930	Tilley.....	..	..	..	....	Removal	.....	.....
1931	Armitage....	M	36	12	....	Removal in 2 operations	.....	Recovery
	Idem.....	F	20	18	....	Removal	Fronto-ethmoid region	Recovery
1931	Terracol and Lamarque	M	18	18	....	Removal	Floor	Recovery
1932	Carmody....	M	26	..	....	Removal	.....	.....
	Idem.....	M	37	34	....	Removal	Posterior wall	Recovery
1932	Grillo.....	F	20	..	....	Removal	Septum	Recovery
1932	Herman and Spiro	M	40	..	....	.....	.....	.....
1932	Parsell.....	M	32	..	....	Removal	Posterior wall	Recovery

TABLE 1.—*Incidence of Osteoma of the Frontal Sinus—Continued*

Date	Author	Sex of Patient	Age	Age at Onset	Trauma	Operation	Attachment	Comment
1932	Roger.....	M	24	..	....	Removal	Fronto-ethmoid region	Recovery
	Idem.....	F	55	54	....	Removal	None	Recovery
1933	Colson.....	M	38	36	....	Removal	Anterior wall	Recovery
1933	di Core.....	M	22	12	....	Partial removal	Posterior wall	Recovery
1933	Fenton.....	F	20	..	....	Removal	.....	Recovery
1933	Knapp.....	F	15	..	....	Adherent to dura; not removed	.....	Recovery
1933	Ninger.....	M	33	30	....	Removal	Posterior wall	Recovery
1934	Baldenweck and others	F	22	20	Yes	None	.....	.....
1934	Causse.....	..	18	..	....	.....	.....	.....
	Idem.....	..	38	..	....	.....	.....	.....
	Idem.....	..	42	..	....	.....	.....	.....
1934	Jeschek.....	M	23	..	....	Partial removal	Anterior wall	Recovery
1934	Neumann....	M	21	..	....	Removal	Fronto-ethmoid region	Recovery
	Idem.....	..	..	..	....	None	.....	Diagnosis by roentgen examination
1934	Patriarca and Rossello	F	75	..	....	Removal	Posterior wall	Recovery
1935	Carmody....	M	27	..	....	Removal	.....	Recovery
	Idem.....	M	35	32	....	Removal	.....	Recovery
	Idem.....	F	13	..	....	Removal	.....	Recovery
	Idem.....	F	42	..	....	Removal	.....	Recovery
1935	Danielewicz..	F	23	21	....	Removal	?	Recovery
1935	Gatewood and Settel	M	32	..	....	Removal	Posterior wall	Recovery
1935	Hoover and Horrax	F	20	19	....	Removal	Posterior wall	Recovery
1935	Kriegsmann..	M	22	..	No	Removal	Fronto-ethmoid region	Recovery
1935	Laskiewicz...	F	14	..	....	Removal in 2 operations	.....	Recovery
1935	del Rossi.....	..	21	..	....	Removal	.....	Recovery
1936	Abbate.....	M	21	..	Yes	Removal	.....	Recovery
1936	Benjamins and Verbeek	M	24	..	Yes	Removal	Septum	Recovery
1936	Jezek.....	M	55	55	....	Removal	Posterior wall	Recovery
1936	di Marzio....	M	24	..	....	Removal	.....	Recovery
	Idem.....	M	27	23	....	Removal	Posterior wall	Recovery
1936	von Matolcsy	M	50	48	....	Removal	Floor	Recovery
	Idem.....	M	21	13	....	Removal	Roof	Recovery
1936	Tinozzi.....	M	15	14	Yes	.....	.....	.....
1937	von Eicken and Schürmann	M	47	17	....	Removal	.....	Recovery
1937	Lejeune-Laoureux	M	37	..	....	Removal	Fronto-ethmoid region	Recovery
1937	Pieri.....	M	32	24	....	Removal in 2 operations	.....	Recovery
1938	Campbell and Gottschalk	M	26	..	....	Removal	.....	Recovery
1938	Devic, Ricard and Mansuy	M	40	..	....	Removal	Floor	Recovery
1938	Ersner and Saltzman	F	19	17	....	Removal	.....	Recovery
1938	Malan.....	M	33	..	....	Removal	Posterior wall	Recovery

TABLE 1.—*Incidence of Osteoma of the Frontal Sinus—Continued*

Date	Author	Sex of Patient	Age	Age at Onset	Trauma	Operation	Attachment	Comment
1938	Pilcher.....	M	20	17	....	Removal	.....	Recovery
1938	Sättler.....	M	19	..	....	None	Septum	Diagnosis by roentgen examination
	Idem.....	M	32	..	....	None	1 to septum 1 to posterior wall	Diagnosis by roentgen examination
	Idem.....	M	58	..	....	None	.....	Diagnosis by roentgen examination
	Idem.....	M	19	17	Yes	None	Fronto-ethmoid region	Diagnosis by roentgen examination
	Idem.....	M	22	..	Yes	None	Floor	Diagnosis by roentgen examination
	Idem.....	M	27	..	Yes	None	.....	Diagnosis by roentgen examination
	Idem.....	M	68	63	....	None	Septum	Diagnosis by roentgen examination
	Idem.....	M	42	..	....	None	Fronto-ethmoid region	Diagnosis by roentgen examination
	Idem.....	M	19	..	....	None	Fronto-ethmoid region	Diagnosis by roentgen examination
	Idem.....	F	38	..	....	None	Septum	Diagnosis by roentgen examination
	Idem.....	M	43	..	....	None	Posterior wall	Diagnosis by roentgen examination
	Idem.....	F	42	..	...	None	.....	Diagnosis by roentgen examination
	Idem.....	F	29	..	....	None	.....	Diagnosis by roentgen examination
	Idem.....	M	42	..	....	None	.....	Diagnosis by roentgen examination
	Idem.....	F	29	..	....	None	Posterior wall	Diagnosis by roentgen examination
	Idem.....	M	41	..	....	None	Posterior wall	Diagnosis by roentgen examination
	Idem.....	F	46	..	....	None	Posterior wall	Diagnosis by roentgen examination
	Idem.....	M	22	..	....	None	Posterior wall	Diagnosis by roentgen examination
	Idem.....	M	34	..	....	None	Roof	Diagnosis by roentgen examination
	Idem.....	M	25	..	....	None	Roof	Diagnosis by roentgen examination
	Idem.....	M	42	..	....	None	Roof	Diagnosis by roentgen examination
	Idem.....	M	49	..	....	None	Roof	Diagnosis by roentgen examination
	Idem.....	F	21	..	....	None	Roof	Diagnosis by roentgen examination
	Idem.....	M	25	..	....	None	Roof wall	Diagnosis by roentgen examination

TABLE 1.—*Incidence of Osteoma of the Frontal Sinus—Continued*

Date	Author	Sex of Patient	Age	Age at Onset	Trauma	Operation	Attachment	Comment
1938	Sattler.....	M	15	..	....	None	Roof	Diagnosis by roentgen examination
	Idem.....	M	25	..	....	None	Septum	Diagnosis by roentgen examination
	Idem.....	M	28	..	....	None	Septum	Diagnosis by roentgen examination
	Idem.....	F	24	..	....	None	Floor	Diagnosis by roentgen examination
	Idem.....	F	24	10	....	Removal	Anterior wall	Recovery
	Idem.....	M	16	16	....	Removal	Lacrimal bone	Recovery
1939	Childrey.....	M	22	..	....	.....	.....	.....
	Idem.....	M	57	..	....	.....	Septum	.....
	Idem.....	F	38	..	....	.....	Floor	.....
	Idem.....	M	40	..	....	.....	Floor	.....
	Idem.....	M	..	..	....	.....	Floor	.....
	Idem.....	F	39	..	....	.....	.....	.....
	Idem.....	M	58	..	....	.....	.....	.....
	Idem.....	M	27	..	....	.....	Floor	.....
	Idem.....	F	58	..	....	.....	.....	.....

Cruveilhier (1835) believed that such a tumor developed between the lamellae of a bone as a "corps osseux enkyste," and, in its growth, pushed the lamellae ahead of it. Virchow (1864) more or less subscribed to the same view and termed the lesion an enostosis. Subsequent histologic examinations have shown, however, that the growth occurs from the cortex and periosteum, not from the medulla. Besides, the septum and the posterior wall of the frontal sinus contain no diploe.

Dolbeau (1871) was the first to question seriously Cruveilhier's view, but he erred in carrying his revolt too far. He stated that exostoses developed by ossification of the mucoperiosteum of the sinuses, entirely independent of the skeleton. Richet (1871), in his critique of Dolbeau's *mémoire,* pointed out that the bone hemorrhage which occurred when Dolbeau removed the tumor refuted his contention and stated that in all such tumors growth was at the expense of the skeleton. Subsequent study has adequately confirmed Richet's view.

Arnold (1873) pointed out that at the junction of the ethmoid and frontal bones two different types of bone, endochondral and membranous, came into contact. On the basis of Cohnheim's general theory that at such points tumors were apt to develop, Arnold said he felt that here was a prime example of such a situation. However, in the present series of cases, in which the attachment of the tumor was mentioned, there were only 26 in which the tumor developed from this region, while in

33 it was attached to the posterior wall, in 23 to the floor, in 18 to the intersinal septum, in 15 to the anterior wall and in 13 to the "roof."

In addition, 8 cases have been reported (table 3) in which the tumor was detached or "dead." In 4 cases suppuration was present, and it is easy to imagine how the pedicle could be destroyed by such a process. In the other 4, however, no infection was present, and an explanation must be sought in a solution of vascular continuity from other causes. Old age, as suggested by Montaz (1888), was certainly not a factor, since all but 1 of the patients in the latter group were under 35. Boenninghaus (1914) suggested trauma as a cause, but the table reveals a history of trauma in only 1 case.

Further evidence against Arnold's view is the fact that a number of cases have been reported in which osteoma developed in several

TABLE 2.—*Age Incidence of Osteoma of the Frontal Sinus by Decades*

Age of patients..	1 to 10	11 to 20	21 to 30	31 to 40	41 to 50	51 to 60	61 to 70	71 to 80
Cases.....	0	63	98	44	24	17	8	1

TABLE 3.—*Data on Eight Cases of Osteoma in Which the Tumor Was Dead*

Date	Author	Age of Patient	Suppuration	Trauma	Comment
1733	Vallisnieri....	..	Yes	...	Pathologic specimen
1885	Tillmanns....	53	Yes	No	Fistula present 6 months
1888	Kikusi.....	45	Yes	...	Fistula drained at intervals for 9 years
1895	Martin.....	23	Yes	...	Fistula present
1898	Tauber.....	32	No	Yes	5 osteomas present, 2 dead
1928	Acker.....	27	No	...	Closure without drainage
1932	Roger.....	55	No	...	Tumor noted only 1 year
1936	di Marzio....	23	No	...	Mucocele present

sinuses, either coincidentally or successively (Golovin, 1914; Haenel, 1904; Rodger, 1928). Also, multiple tumors have occurred in different locations in the same frontal sinus (Tauber, 1898).

Bornhaupt (1881) expressed the belief that remains of the chondrocranium could give rise to bony tumors, but there is not a single histologic report, with the exception of that of Virchow (1864), in which persistence of cartilage in the tumor has been seen in the frontal sinus. Moure and Nadal (cited by Causse, 1934), however, noted 1 case of osteochondroma in the ethmoid. Consequently, no importance can be attached to this theory, which is, essentially, an adaptation of von Rokitansky's general theory that exostoses are ossified chondromas.

Fetissof (1929) stated that osteoma develops from a nest of periosteum split off at an early age, in the presence of irritation, such as trauma or infection. He further stated (1931) that this could occur in

two ways: (1) osteoblastically, at the expense of the periosteal osteoblasts, and (2) by metaplasia of fibrous tissue into bone, osteoclasis going hand in hand with osteoblasis.

This is the most promising theory to date, but it still lacks much in order to explain all of the observed facts. Fetissof, and also Pieri (1937), felt that some further factor, such as trauma or infection, would be necessary to initiate growth, but neither of these factors has been present in all of the cases reported. In the present series there was a history of trauma in only 37 cases, while in 32 cases it was definitely stated that there was no such history. In the remaining cases no mention was made. It is possible, however, that trauma had occurred and had been forgotten.

As to infection, which was held to be important by Gerber (1907) and by Eagleton (1935), there was evidence of such a complication in only 31 cases. Pfeiffer (1912) pointed out that osteophytes may result from infection, but these must be differentiated from osteomas. Ersner and Saltzman (1938) found Staphylococcus albus haemolyticus in their case and theorized that a low grade infection might stimulate osteogenesis more than osteoclasis. This would not apply, obviously, to those cases in which no infection is present and in which the history suggests there has been none in the past. In former years, syphilis was held to be a causative factor of osteoma, but it is no longer believed to be such. Knapp (1933) has suggested, without proof, that endocrine changes might play a role.

Summing up the whole question of etiology, it seems to me that Mitvalsky (1894) expressed it succinctly when he said:

> "Quant a moi, je crois qu'il vaut mieux, quant a l'origin de nos osteomes, prononcer franchement le mot 'ignoramus,' que de se tromper les uns les autres par des suppositions imaginaires." (Personally, I believe it is better, as regards the origin of osteomas, to admit frankly that we do not know, rather than to waste time in theoretic speculations.)

The pathologic appearance of osteoma has been described in detail by Eckert-Möbius (1929), Fetissof (1929), Causse (1934) and Malan (1938), so that I shall not discuss it further here except to list the main types of osteoma seen. The first is the eburnated type, in which there is a total absence of haversian canals. The second is the compact osteoma, also a hard tumor, in which the haversian canals are small and lie in all directions. The third type is the spongy osteoma, which is softer than either of the other two and consists of bony trabeculae with intervening marrow spaces. The fourth is the mixed type, in which the preponderance of type of tissue depends somewhat on the age of the tumor. Causse (1934) also mentioned chondro-osteoma, found in the ethmoid sinus, and angio-osteoma, found in the sphenoid sinus. Stout (1932) pointed out the resemblance of osteoma to the rapidly pro-

liferating bone found in the repair of fractures. In 2 cases (Amussat, 1834; Babcock, 1924) connective tissue developed rather extensively, so that a softer type of tumor appeared. In 2 cases (Beck, 1930; von Eicken and Schürmann, 1937) the tumor was made up of an osteoid type of tissue.

Before the advent of the roentgen ray the diagnosis of osteoma was practically impossible until pressure symptoms in eye, nose or brain became apparent. Consequently, treatment was rendered more difficult and the outcome less assured. The first case in which roentgen examination was used for diagnosis was that of Coppez in 1899. Since that time its use has been routine.

The differential diagnosis, as pointed out by Dahmann (1922), includes consideration of orbital lipoma, cyst, atheroma and teratoma;

TABLE 4.—*Reports of Pyocele as a Complication of Osteoma of the Frontal Sinus*

Date	Author	Date	Author
1758	Sporing	1883	Weinlechner
1841	Bouyer	1886	Lucas-Championnière
1845	Adelmann	1888	Kikusi (case 2)
1865	Textor	1895	Coppez (case 2)
1881	Bornhaupt	1895	Martin
1882	Imre	1899	de Santi
1875	Burow	1900	Witzheller
1907	Gerber (case 2)	1929	Rosenstein
1914	Zumhasch	1930	Brown
1915	Lack	1930	Leavenworth
1919	Jacques	1934	Jeschek
1924	Faulkner	1936	Jezek
1927	Harper	1936	di Marzio (case 4)
1927	Mouret and Dejean	1936	von Matolcsy
1927	Riecke	1937	Lejenune-Laoureux
1928	Rodger		

mucocele, pyomucocele, hematoma, hygroma, chondroma, fibroma, sarcoma, fibrosarcoma, carcinoma, cholesteatoma and osteomyelitis.

COMPLICATIONS

Gerber (1907) pointed out that, like nasopharyngeal fibromas, osteomas are benign histologically, but malignant clinically ("Histologiquement elles sont benignes et cliniquement elles sont malignes"). Their position among highly important structures gives them extensive opportunity for serious damage.

Growth into the region of the nasofrontal duct may isolate the sinus, with the production of a pyocele, pyomucocele or mucocele. Of the first, there were 31 cases in the present series (table 4). In 3 cases (Hamilton, 1901; Claus, 1928; von Eicken and Schürmann, 1937) a pyomucocele was diagnosed.

Mucocele was present in 40 cases (table 5). Growth in the direction of the walls of the sinuses produces resorption of the bone and, eventually, perforation.

Growth into the ethmoid or maxillary sinuses may cause infection or mucocele there, and growth into the nose may produce obstruction to breathing. The latter symptom, however, is more common in cases of ethmoidal osteoma.

Extension into the orbit causes, first, a palpable tumor in the superointernal angle, and later, a protrusion of the eye forward, downward and outward. While an ethmoidal osteoma may produce the same displacement, it usually pushes the eye forward and outward only. In Fridenberg's case, however, the tumor grew down from the supraorbital ridge in such a way that it caused enophthalmos. A maxillary osteoma usually pushes the eye up and out. In several cases in the present series the eye had been entirely destroyed.

TABLE 5.—*Reports of Mucocele as a Complication of Osteoma of the Frontal Sinus*

Date	Author	Date	Author
1871	Birkett	1898	von Navratil
1871	Richet	1898	Tauber
1882	Teillais	1899	Luc
1892	Maydl (also reported by Mitvalsky, 1894)	1900	Zimmermann
1892	Poppert	1903	Hollet
1894	Mitvalsky (case 2)	1904	Axenfeld (also reported by Hucklenbroich, 1905)
1896	Coppez	1905	Taylor
1896	Sattler	1908	Selmemi
1910	Manasse	1927	Neumann
1911	Dubar and Potel	1929	Browder
1912	Pfeiffer	1929	Howarth
1916	Chapman	1930	Beck
1924	Howarth	1931	Armitage
1924	Versteegh	1932	Grillo
1924	Vulowitsch	1933	di Core
1925	Bacher	1933	Knapp
1925	Buys	1934	Patriarca and Rossello
1926	Lillie and Anderson	1938	Campbell and Gottschalk
1927	Caccialupi	1938	Devic, Ricard and Mansuy
1927	Garretson	1938	Ersner and Saltzman

Extension into the cranial cavity may produce one of several complications. Mental disorder, epilepsy and vertigo were seen but rarely, considering the large number of cases in which intracerebral extension occurred. Cerebrospinal rhinorrhea was reported in 1 case (Berg, 1889). Discharge and headaches stopped after operation. Meningitis, except in 1 case (Mackenty, 1918), occurred only as a complication due to surgical treatment. Brain abscess was noted at autopsy in 9 cases. In Schlesinger's case (1913) no operation was done, and death was attributed to glioma. The most interesting complication was cranial pneumatocele, which was reported eight times. The air was extradural in 2 cases (Carmody, 1932; Benjamins and Verbeek, 1936); subdural, extracerebral in 4 cases (Tyczka and Chorobski, 1929; Herman and Spiro, 1932; Baldenweck and others, 1934; Devic and Mansuy 1938), and ventricular in 2 cases (Armitage, 1931; Campbell and Gottschalk, 1938). Echlin (1934) stated that osteoma of the cranial surface may

arise secondarily from a fibroblastoma, but such a coincidence was not noted in this series.

Treatment has always been difficult, but in the preantiseptic era it was especially hazardous. Some hardy surgeons boldly attacked the tumor with chisel, hammer and osteotome, while others preferred to apply caustics and await gradual exfoliation. In 1 case (Hilton, 1836) the tumor became detached spontaneously and dropped off. This outcome was considered ideal.

While MacKenzie (1833), Middlemore (1835) and Dolbeau (1865) blandly advised the surgical removal of osteomas, Berlin (1880) pon-

TABLE 6.—*Mortality in Cases of Osteoma Reported Since 1875 in Which Surgical Treatment Was Used*

Date	Author	Sex and Age	Operation	Cause of Death
1882	Tweedy	M 25	Tumor attacked with chisels and drills. Orbital portion broken off	Basilar meningitis 1 month after operation
1883	Weinlechner	M 51	Osteoma removed from right frontal sinus. Healing. Later, osteoma and polyps removed from nose	Erysipelas followed last procedure, and patient died
1883	Panas	F 18	2 prior unsuccessful operations followed by fistula. At third attempt, only projections of tumor removed	Meningitis. Tumor extended into frontal lobe. Suppurative phlebitis of veins from operative field
1905	Taylor	M 16	Complete removal of tumor. Dura exposed	Meningitis 1 week postoperatively
1918	Boardman and Donovan	M Young	Complete removal	Meningitis
1918	Culbert	M 43	Dura torn; operation stopped. 6 months later, intranasal procedure. Piece extending into brain left intact	Patient died 3 months after last procedure. Several large abscesses in frontal lobes. Patient also had syphilis
1927	Neumann	F Old	Removal of tumor which had exposed dura	Frontal lobe abscess

dered a 25 per cent mortality and advised strongly against operative intervention. He suggested enucleation of the eye when it appeared that the eye could no longer be saved. Even as late as 1918, Mackenty stated that the consensus was that removal is so difficult that these tumors might be considered inoperable. Berlin's figures included all types of orbital exostosis, but in reviewing the present series prior to 1875 (arbitrarily taken as the beginning of the antiseptic-aseptic era) one finds 32 cases in which operation was performed, with 10 deaths—a mortality of 31.2 per cent. Under these circumstances, Berlin was certainly justified in his conservative point of view. Since 1875, there have been 189 cases in which surgical treatment was used, with 7 deaths, a mortality of 3.7 per cent (table 6). The total mortality for the entire group of surgical cases is 7.76 per cent.

Most of the older surgeons attacked the tumor directly with chisels and saws, but Andrews (1887) advised removing enough bone from

around the tumor to permit it to be easily extricated. The Killian, Moure or inverted T incision has been used. In 1919 Lang and Armour reported using the osteoplastic flap of Cushing in a case in which intracranial extension had occurred. This procedure has also been used by Armitage (1931), Hoover and Horrax (1935), Benjamins and Verbeek (1936), Campbell and Gottschalk (1938), Malan (1938) and by Pilcher (1938). Hempstead (1938) pointed out with reason, however, that this method is illogical in cases in which the dura or cribriform plate is not involved.

In several cases, recurrence of the tumor was mentioned. Since most of the patients, however, were not observed over a sufficiently long period, it would be impossible to arrive at a definite conclusion on this point.

Although exposure of the dura has not appeared to be a highly dangerous procedure, di Core (1933) left a portion of the osteoma attached to the posterior wall, preferring, apparently, to risk recurrence rather than to expose the dura. Most surgeons, I believe, would have removed the entire neoplasm.

Opinion as to the time of operative intervention is sharply divided. Roger (1932) and Causse (1934) represent the French school, which advocates complete and early extirpation as soon as the diagnosis is made. Lillie (1938) expressed the opinion of many American and English writers, who consider surgical intervention unnecessary unless definite symptoms and signs demand it. Beck (1930) made the unsupported statement that 48 per cent of the patients with osteoma for whom operative treatment is not used die of complications.

In view of the well known tendency of osteomas to grow, it would seem more logical to me to remove them before they involve important neighboring structures. If they are small when first seen, one can follow their development with roentgen examinations, and if they are observed approaching the orbit or the cranial walls of the sinus, surgical intervention should be advised. If the patient then refuses operation, the responsibility rests on him.

Some of the fatalities in the past have been due, I am sure, to surgical intervention during an acute or subacute infection. Certainly one should withhold operation until infection has subsided, since the field of operation is so proximate to the dura and the brain. If the organism present is a streptococcus or a pneumococcus, the patient should be saturated with sulfanilamide preoperatively.

SUMMARY

Two cases of osteoma primary in the frontal sinus are reported, several interesting historical cases are reviewed and a reproduction of the first illustration of a patient who had had osteoma is shown.

A survey of the literature revealed 321 cases, a figure which indicates that osteoma, while not common, certainly is not rare. About 40 per cent of all osteomas of the accessory sinuses originate in the frontal sinus. The disease is essentially one of youth, by far the greatest incidence occurring in the second and third decades. It also attacks males more frequently than females. The various theories as to its causes are reviewed, the conclusion being reached that no completely satisfactory explanation has as yet been devised. The pathology is reviewed briefly, four main types of osteoma being described. In connection with the diagnosis of osteoma it is pointed out that the first case to be recognized by roentgen examination was reported by Coppez in 1899. The complications caused by the growth of the neoplasm into the nasofrontal duct, the neighboring sinuses, the orbit and the cranial cavity are discussed as illustrative of Gerber's statement that osteomas are benign histologically but malignant clinically. Treatment is then discussed, and statistics are shown, indicating that in the preantiseptic era the surgical mortality was 31.2 per cent, while since 1875 it has been only 3.7 per cent. The opinion is expressed that operation should be carried out while the tumor is small, rather than after the appearance of symptoms of extrasinal extension. If infection is present, the resort to surgery should be delayed until the infection is quiescent.

Since this paper was prepared for publication, articles describing 14 additional cases have been noted (Proetz; Smith; Handousa).

Miss Sue Biethan, medical librarian of the University of Michigan, and her assistants and Miss Frances Hannum and her assistants, of the Ann Arbor Public Library, made available the works listed in the bibliography. Material was also obtained from the following libraries: John Crerar Library, Chicago; Quine Library, Colleges of Medicine and Dentistry, University of Illinois, Chicago; Bio-Medical Libraries of the University of Chicago, Chicago; Boston Medical Library, Boston; Library of the Surgeon General's Office, Washington; New York Academy of Medicine Library, New York; Medical Library, Columbia University, New York; American Museum of Natural History, New York; Lane Medical Library of Stanford University, San Francisco; Washington University School of Medicine Library, St. Louis; Harvard University, Schools of Medicine and Public Health Library, Boston; William H. Welch Medical Library, Baltimore; Library of College of Physicians of Philadelphia; American College of Surgeons Library, Chicago.

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SURGICAL TREATMENT OF BILATERAL PARALYSIS OF THE ABDUCTOR MUSCLES

JOSEPH D. KELLY, M.D.
NEW YORK

It is the purpose of this paper not to discuss at length the symptoms, diagnosis and causes of bilateral paralysis of the abductor muscles of the larynx but to cover in a general way the surgical treatment and the results obtained by the various operations. In other words, it is an attempt to answer the question—"What can I do to relieve the patient suffering with bilateral paralysis of the abductor muscles, and what results can I expect?"

In October 1939, in one of the public hospitals in New York, I was confronted with a patient who was suffering from this condition after several operations on her thyroid gland. She wanted something done to relieve her of the tracheotomy tube, and she wanted it done quickly, because of the distressing annoyance caused by this plumbing in her trachea. I assured her that something would be done at the first opportunity. Up to this time my one and only resection of a vocal cord had been a failure, and on consulting the literature I found that there had been few successful operations performed and reported. Either the patient had an airway adequate for his normal requirements and the voice was very poor, or the voice remained good and the patient could not be decannulated. After study, I concluded that the problem of bilateral paralysis of the abductor muscles might be considered in the following manner:

Bilateral paralysis of the abductor muscles is of two types, that which comes on gradually as a result of injury to the recurrent nerves or of a lesion or an infection of the central nervous system and which may be borne by the patient without succumbing to tracheotomy and, the second type, that which is ushered in with a violent spasm of the conus muscularis, causing a most distressing dyspnea and relieved only by a tracheotomy. Patients with the first type, even though they are incapacitated and suffer much distress from periods of dyspnea, abhor the idea of a tracheotomy. Those with paralysis of the second type, although they welcome tracheotomy as a life-saving measure, soon tire of the tracheotomy tube, which robs them of their voice and irritates their trachea.

Read before the Scientific Section of the American Academy of Ophthalmology and Otolaryngology, Cleveland, Oct. 9, 1940.

Even though the laryngologist is able to supply them with the ingenious Tucker tube or some other bivalve tube to strengthen their voice, they will ask to be relieved of the annoying cough and tracheal reflexes caused by the tracheotomy tube. Hence, all of these patients look for an operation which will restore the function of the larynx as a breathing tube and will preserve a pleasant, useful voice.

To this end several groups of operative procedures have been devised. The operations of the first group, and perhaps the ideal procedures, were those in which an attempt was made to suture the ends of the severed nerve,[1] or to anastomose it with one of the nerves in the neck. These operations were not successful, because the ideal is difficult to obtain and because it was found that the impulses arriving as a result of suture or anastomosis went not only to the abductor but also to the adductor muscles, producing spasmodic movements of the cords, and left the patient as distressed as he was before the operation. The operations also had a limited use, because they could be employed only in those cases in which the surgeon was positive that the nerves had been cut and that he would be able to pick up one or more severed ends. Ballance, Frazier and Lahey were the pioneers who attempted to accomplish this ideal.

The operations of the second group were directed at the interior of the larynx and at the paralyzed cords themselves. It seemed a logical thing that if the surgeon could eliminate one cord the patient would have more breathing space, and with the other cord in the midline there was hope that he would have a satisfactory voice. Hence, the development of cordectomy. This operation was tried and found a failure, because scar tissue readily formed a new cord and often caused more distress to the patient than he had suffered before the operation. Some laryngologists [2] at this time felt that the removal of more tissue would bring the cicatrices closer to the internal surface of the thyroid cartilage, thus leaving a larger aperture in the larynx. It was thought that if the ventricle and the cord were removed at the same time the operation would be a success. However, this procedure failed too. It was followed by the formation of scar tissue and in some instances by cicatricial stenosis. It had many weaknesses, it was inaccurate and it neglected any consideration of the voice-producing tissues of the larynx. It was not a surgical procedure; it was just a punch in the dark.

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2. Jackson, C.: Ventriculocordectomy: A New Operation for the Cure of Goitrous Paralytic Laryngeal Stenosis, Arch. Surg. **4**:257-274 (March) 1922.

Dr. Hoover [3] realized that the failure of all these procedures was due to the formation of intralaryngeal scar tissue, and he tried to overcome this difficulty by submucous resection of the cord and adjacent tissues following laryngofissure. This operation was a step in the right direction. The procedure has proved successful in the hands of Dr. Hoover, but it has been a failure in those of many others, including my own. I cannot but praise the operation; it was with this method that laryngologists had their first success. I find that Dr. Hoover [4] has reported satisfactory conclusions in 75 per cent of the cases in which he has operated but has admitted that the patients have a poor voice. A few other operators have reported good results with thic technic; however, the submucous resection is difficult,[5] and I believe that it was with the idea

TABLE 1.—*Ventriculocordectomy and Cordectomy—Results Obtained by Various Operators*

Surgeon	No. of Cases	Results		Complications	Comments
		Voice	Airway		
			Ventriculocordectomy		
Figi	1	Not stated	Inadequate	None	None
Hitz	1	Worse	Inadequate	None	"Tried operation twice"
Stokes	9	Worse 2 Unimproved 7	Inadequate 9	Hemorrhage ? Synechia ? Mediastinitis 1	"Operation unsatisfactory"
Clerf	5	Worse 5	Inadequate 5	None	"In none of these were the results satisfactory, for the voice was seriously impaired; in addition, there was ultimate return of much of dyspnea due to cicatricial changes"
			Cordectomy		
Birkett	1	Worse	Inadequate	None	"No improvement"
Howell	1	Improved	Inadequate	None	"Not a success"

of simplifying this part of the Hoover operation that Dr. Loré,[6] of New York, came forward with his method of elevating the mucous membrane over the area in which Hoover did a submucous resection. By elevating the mucous membrane, it was possible to come down directly on the tissue to be removed; hemorrhage was more readily controlled, and a more

3. Hoover, W. B.: Bilateral Abductor Paralysis: Operative Treatment by Submucous Resection of the Vocal Cords, Arch. Otolaryng. **15**:339-355 (March) 1932.

4. Lahey, F., and Hoover, W. B.: Injury to Recurrent Laryngeal Nerve in Thyroid Operations, Ann. Surg. **108**:545, 1938.

5. Zinn, W. F.: Bilateral Abductor Paralysis, Ann. Otol., Rhin. & Laryng. **44**:164 (March) 1935.

6. Loré, J. M.: A Suggested Operative Procedure for the Relief of Stenosis in Double Abductor Paralysis: An Anatomic Study, Ann. Otol., Rhin. & Laryng. **45**:679-686 (Sept.) 1936.

complete removal of the tissue could be made under direct vision. Loré went a step further in his proposed operation, and instead of just removing a part of the vocal process of the arytenoid cartilage, he removed it completely. This part of his procedure, I think, spelled success for his method, because the greatest number of successful results for the relief of paralysis of the abductor muscles has been obtained by those operations which had something to do with the arytenoid cartilage. However, Loré was not the first surgeon to think of removing the arytenoid cartilage as a help in relieving this condition. Dr. Charles H. Baker,[7] of Bay City, Mich., reported the case of a 9 year old boy on whom he operated in 1916, removing the right cord and arytenoid

TABLE 2.—*Submucous Resection of Cord and Vocal Process of Arytenoid Cartilage—Hoover Technic*

Surgeon	No. of Cases	Results		Complications	Comments
		Voice	Airway		
Stevenson	1	Improved	Adequate	Hemorrhage	"Satisfactory breathing"
Kistner	2	Unimproved	Adequate	None	None
Tobey	1	Unimproved	Adequate	None	"Submucous of the cord and fixation to thyroid cartilage with catgut"
Beyer	1	Improved	Inadequate	None	"This patient was not decannulated because he lived in the country"
Hoover	8	Worse 8	Adequate 6 Inadequate 2	Infection 1 Unknown 7	"The operation sacrifices the voice in a large measure. We feel that the 75% excellent results can still be further improved"
Stokes	4	Unimproved	Not stated	Not stated	"In my own small personal series (13 cases) I favor the laryngofissure with unilateral complete excision of vocal cord, ventricular band and vocal process"

cartilage following laryngofissure, with an excellent result. In view of the result obtained by this physician and recorded in his published paper, it seems that laryngologists neglected something by waiting until 1932 for Dr. Hoover to start them thinking again. The only reason I can advance for their delay in accepting Dr. Baker's technic is that they were afraid of removing too much tissue from the larynx, which would be tragic, and that his was the only case reported up to that time.

In 1939 a man came over the mountains from the West, a man who had been trained in orthopedics, and at once began to tell laryngologists that they had missed the boat completely—that there was no need for intralaryngeal surgical procedure and that the solution of their problem was the same as that of the problem of a paralytic ankylosis

7. Baker, C. H.: Report of a Case of Abductor Paralysis with Removal of One Vocal Cord, J. Michigan M. Soc. **15**:485 (Oct.) 1916.

of a joint in any other part of the body. He utilized the principle of substituting a viable muscle for a paralyzed one and in this particular instance attached the anterior belly of the omohyoid muscle to the muscular process of the arytenoid cartilage and began to report successful results. This procedure appealed to every laryngologist confronted with a case of paralysis of the abductor muscles. A number of operations were performed by enthusiastic laryngologists, but reports of successes were few. Men began to doubt the value of the method when the patients went on for two or three months without any improvement. Such was the position of the King [8] operation when I was trying to find some fool-proof method for use in the case of double paralysis of the abductor muscles in my service. However, the operation appealed to me because: First, it was extralaryngeal, thus avoiding the formation

TABLE 3.—*Submucous Resection of Cord and Arytenoid Cartilage Through Laryngofissure—Loré Technic*

Surgeon	No. of Cases	Results		Complications	Comments
		Voice	Airway		
Rawlins	1	Worse	Adequate	None	"Perfect result except for voice. Only necessary to operate on one side to get good result"
Johnson	1	Improved	Adequate	None	"Voice is better than in those who have had King operation done. Removal of arytenoid is most important step"
Baker	1	Good	Adequate	None	Operation was performed in 1916
Woodward	1	Worse	Inadequate	None	"A very unsatisfactory procedure; operation was carefully and completely done"

of scar tissue; second, it preserved the tissue of the larynx, thus increasing the chances of the patient's having a good voice, and third, it was directed toward the arytenoid cartilage, which was important to me at the time. I could not see the virtue of trying to attach the omohyoid muscle to the arytenoid cartilage, because I felt that the best one could expect would be that the muscle would attach itself to the surrounding tissues and not to the cartilage. I felt that if the arytenoid cartilage was removed by an extralaryngeal route the result would be good. Since reporting his first case, Dr. King [9] has improved on his technic by the simple expedient of completely mobilizing the arytenoid cartilage by cutting it practically free from the articular process of the cricoid

8. King, B. T.: A New and Function-Restoring Operation for Bilateral Abductor Cord Paralysis: Preliminary Report, J. A. M. A. **112**:814 (March 4) 1939.

9. King, B. T.: A New and Function-Restoring Operation for Bilateral Abductor Cord Paralysis, Tr. Am. Laryng. A. **61**:264, 1939.

cartilage. At this point I will let Dr. King tell his own story in the way of a personal communication received from him in answer to one of my questionnaires, under date of July 31, 1940.

I have now operated on 24 patients for bilateral abductor cord paralysis, 22 women and 2 men. Their ages are from 26 to 73. There were no deaths. Complications were: infections in 6 cases and chronic tetany in 1 (the patient gave a history of chronic tetany at the time of operation for goiter). Twenty-two patients had had thyroidectomy and 2 had infections of the larynx, 1 tuberculous and 1, a child 1½ years of age, probably streptococcic.

Simple transposition of the omohyoid muscle was done in 3 instances. In 21 cases there has been a thorough division of the cricoarytenoid joint capsule and

TABLE 4.—*King Operation—Results Obtained by Various Operators*

Surgeon	No. of Cases	Results		Complications	Comments
		Voice	Airway		
Figi	1	Not stated	Inadequate	None	"Patient able to breathe freely with cannula corked 2½ months following operation, but has not yet been decannulated"
Knight	1	Unimproved	Inadequate	None	"About 30 per cent improvement in abduction of right cord; not sufficient for decannulation"
Rives	1	Unimproved	Inadequate	None	"Operation failed because muscles and fascia of intra-arytenoid structures were not severed. There was motion of left arytenoid following operation"
Clerf	3	Normal 3	Inadequate	Skin infection 1	"In two cases, there was motility and increase in the airway so that patient could wear the cannula blocked. In the third case, one month after operation, there is no motion of larynx and patient has not been decannulated"
King	24	Good 22 Poor 2	Adequate 24	Infection 6	"Three cases had simple transposition of omohyoid muscle. Twenty-one cases had complete division of cricoarytenoid capsule and outward displacement of the arytenoid cartilage"

outward displacement of the cartilage. In 23 cases the omohyoid muscle was transposed, and in 1 it was not. (In this case the patient secured an excellent airway and a fairly good voice and gained 32 pounds [14.5 Kg.]; paralysis had existed for eighteen years.) In 2 cases the lower half of the arytenoid cartilage was removed, though this had not been planned as part of the operation; the cartilage was fractured in attempting to pass a suture through it, and the lower half was so separated from surrounding structures that it was simply removed, as no circulation could possibly have been left to supply it. In both these cases, the patients secured good results in both airway and voice. However, the airway in these 2 patients was not as great as that in a number of others. In other words, the outward displacement of the vocal cord was not as much as when the cord is pulled outward by displacing the arytenoid cartilage outward.

I am not at all sure that all of these patients would not have gotten along just as well with outward displacement of the arytenoid cartilage as with both displacement and transposition of the omohyoid muscle. When the original article was published, I attached more importance to transposition of the muscle than at

the present time, and it may be that I shall dispose of transposing the omohyoid muscle. I have found it important to examine the larynx of the patients during operation, to determine if sufficient airway had been gained.

From this communication it will be observed that Dr. King began to get his greatest number of successful results when he completely mobilized and displaced outward the arytenoid cartilage, that he is now of the opinion that the transplantation of the omohyoid muscle is of little importance in the success of his operation and that in the future he will most likely confine his efforts to the arytenoid cartilage.

It was necessary that something be done to satisfy and relieve the patient mentioned early in this paper, and I could not determine which type of operation offered me the greatest chance of success. The immediate consideration was that this patient refused to be operated on if there was a possibility of losing her voice. To assure the retention of voice, it would be necessary to develop some surgical procedure that would be as nearly extralaryngeal as possible, to preclude the possibility of intralaryngeal contractions which might deprive her of a satisfactory voice. The conclusion of my study was that if I could perform an arytenoidectomy through the wing of the thyroid cartilage I might accomplish three things:

1. I should destroy the tension of the cord on the side on which the operation was performed, which is caused by the action of the cricothyroid muscle, and allow the cord to fall away from the midline of the larynx.

2. The space created by the loss of the arytenoid cartilage would be obliterated by nature and thereby cause an atrophy of the muscles on that side, which would further increase the area between the cords posteriorly.

3. The position of the anterior third of the cord would be slightly altered in relation to the side which was not operated on, and in that way I should preserve, to a reasonable extent, a proximity of her cords which would give her a fairly serviceable voice.

It was with these ideas in mind that my associate, Dr. John F. Daly, and I did some work in the morgue to determine the easiest route for extracting the arytenoid cartilage and the feasibility of doing so. In studying the larynx, we found that if a window were made in the lower posterior third of the thyroid cartilage, with its anterior border limited by a line separating the middle and the posterior inferior third of the thyroid cartilage, we should come down on the muscular structure covering the arytenoid cartilage and that, by careful dissection, the arytenoid cartilage could be removed with little damage to the intralaryngeal mucous membrane. We also found, by examining the larynx after this procedure had been performed, that the cord on the side of operation fell away from the median line and assumed a lower level than the cord

on the side on which the operation had not been done and that if nature followed the usual course in repairing tissue by obliterating cavities the contraction of the cavity left by the removal of the arytenoid cartilage would give the patient a satisfactory airway.

Previous to the operation we were undecided as to what method of anesthesia we should use. It was impossible to remove the tracheotomy tube from this patient for more than half a minute, because of the peculiar conformation of the scar tissue in the skin surrounding her tracheotomy opening. As soon as the tube was removed, the scar tissue contracted and the patient was unable to inspire any air through the opening, and she was unable to, inspire through her larynx, because the cords were overlapping each other. There were also contractions of the old thyroidectomy scars which prevented the patient from lifting her chin. If she did raise her chin, the tracheotomy opening became tighter as a result of the tension on the scar tissue. Therefore, it was decided that the first operation should be performed under general anesthesia, in order that any unforeseen eventualities might be handled quietly and deliberately. We therefore enlisted the services of Dr. Paluel Flagg, our anesthetist, and together worked out a plan with him to give the patient some preoperative medication and to have the administration of the anesthetic started through the tracheotomy tube. When the patient was sufficiently anesthetized, Dr. Flagg was to endeavor to pass one of his intratracheal tubes through the cords into the trachea, and we would then be able to remove the tracheotomy tube and the patient would be in an ideal condition for us to proceed, without the hindrance of tension. This procedure was carried out, and everything progressed as we had planned. The scar contractures of the neck were corrected, the larynx exposed on the left side, the window made at the selected position in the thyroid cartilage and the arytenoidectomy carried out as it had been done on the cadaver. Our only trouble was that we encountered a sharp little bleeder in dissecting the muscle tissue away from the posterior surface of the arytenoid cartilage. It required the administration of a little epinephrine hydrochloride and the application of pressure to control the bleeding; otherwise the operation was uneventful. The skin of the neck was closed and a puncture wound drainage opening made in the skin over the thyroid cartilage, through which a cigaret drain was placed and left in position for five days. A stomach tube was then inserted through the nose and left in position for the same length of time. The tracheotomy tube was replaced, and at the end of about one week or ten days the patient began to breathe through her larynx.

The operation was performed on Nov. 3, 1939, and the patient was discharged from the hospital on November 15, at which time she was wearing a stopper in her tracheotomy tube twenty-four hours a day. My associate and I could see the improvement taking place in her larynx

from November 15 until she was sent back to the hospital, January 4, for removal of the tube. The tube was removed on the day she entered the hospital, and she was allowed to leave the hospital on January 10 and has since carried on her duties as housewife with little inconvenience.

Since performing the first operation, I have felt that this procedure could be used with the patient under local anesthesia, with safety and with little discomfort to the patient. My associate and I had our opportunity of doing this operation with the patient under local anesthesia on Feb. 19, 1940, when a case was turned over to us by the broncho-

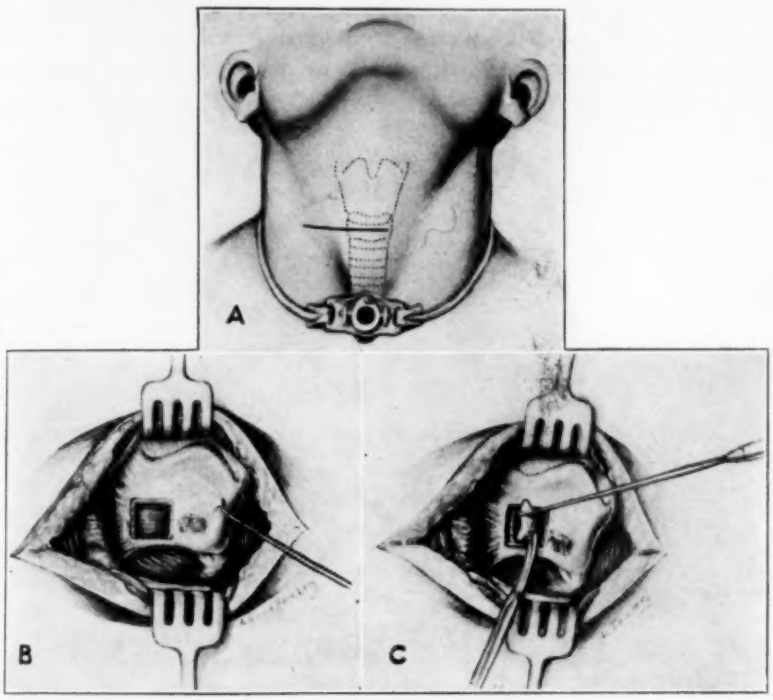

Fig. 1.—*A*, location of the incision in the second operation; *B*, exposure of muscle tissue over the arytenoid cartilage, and *C*, dissection of the arytenoid cartilage through muscle tissue.

scopic department. The patient was a man 68 years of age, who suffered with bilateral paralysis of the abductor muscles and who had been wearing a tracheotomy tube for almost a year. He was anxious to have something done, because of the severe coughing spells and irritation experienced on retiring. He felt that his tracheotomy tube was responsible for his spasms, because when it was removed he obtained definite relief.

It was remarkable to me how little interference with deglutition was caused by removal of the arytenoid cartilage with the patient under local anesthesia. As soon as we finished the operation, the patient sat up on the table and asked for a drink of water. He took his drink without any evidence of difficulty or distress in swallowing. Because of this action, he was allowed to remain on a full diet with no restrictions during the period of his convalescence.

One thing we noticed in doing this second operation was that we experienced more difficulty in removing the arytenoid cartilage than in the first case. This, I believe, was due to the fact that in the first operation, in which the anesthetic was given by intratracheal tube, the tube helped to fix the arytenoid cartilage in one position and acted as a point of counter pressure in cutting the arytenoid cartilage free from the

Fig. 2.—View of the larynx two months after operation, showing the position of the right cord.

articular surface of the cricoid. In the future I shall have a bronchoscope passed when the operation is to be performed with the patient under local anesthesia. We used a half Kocher incision in this case for exposing the right side of the larynx. The incision used by Dr. King would be serviceable and does not require the cutting of any pretracheal muscles, but I believe that the half Kocher incision gives a better scar.

This second case of ours terminated tragically. The patient died within seven weeks after his operation. He had a cardiorenal involvement in an advanced stage and his blood pressure was 200 and over during his stay in the hospital following operation. The tracheotomy tube was removed five weeks after his operation, and he was allowed to return to his home. He remained at home for about a week and was brought back to the hospital by his daughter, who said that he was having difficulty in breathing. Examination of the larynx at this time showed

that the patient had ample airway, in fact, more than the patient whose operation was performed earlier. However, he was nervous and had a rapid pulse and blood pressure of over 220. A quarter grain of morphine sulfate was given hypodermically. The patient became quiet and remained so until midnight, when he again required morphine sulfate. The following day my associate and I reopened the tracheotomy wound and inserted a tube, and during the patient's remaining five days in the hospital his tube was corked. He died of cardiac thrombosis a week or so after leaving the hospital the second time.

With these 2 cases as a starter and with my interest in this type of operation aroused, I gladly consented when Dr. Myers asked me if I would read a paper at this meeting of the American Academy of Ophthal-

TABLE 5.—*Compilation of Results Obtained by Various Operative Procedures*

Operations	No. of Cases	Etiology	Tracheotomy Done in	Results		Failures
				Voice	Airway	
Ventriculocordectomy	16	Thyroidectomy 14 Tumor of neck 1 Syphilis 1	16	Poor 16	Adequate 0	16
Intraoral cordectomy	2	Thyroidectomy 2	2	Improved 1 Worse 1	Adequate 0	2
Submucous cordectomy (Hoover)	17	Thyroidectomy 16 Diphtheria 1	Unknown	Improved 2 Unimproved 15	Adequate 10 Not stated 4 Inadequate 3	3
Submucous cordectomy and arytenoidectomy (Loré)	4	Thyroidectomy 2 Diphtheria 2	2	Improved 2 Worse 2	Adequate 3	1
King operation	30	Thyroidectomy 28 Infection 2	Unknown	Improved 27 Poor 3	Adequate 24	6
Arytenoidectomy extralaryngeal	2	Thyroidectomy 1 Central lesion 1	2	Improved 2	Adequate 1	1

It will be noted in this table that the greatest number of successes were obtained in those operations which prevented the formation of scar tissue and either removed the arytenoid cartilage or completely mobilized it so that it could be fixed in abduction.

mology and Otolaryngology. I did so for three reasons: First, I thought that the literature on the question of the surgical treatment of bilateral paralysis of the abductor muscles was confusing. Of course, this was before Dr. King's report on his 24 cases. Secondly, I felt that I might be rendering a service to the society if I could get enough laryngologists to tell me their experiences with cases of this type and whether or not they had done any operation other than tracheotomy. I felt that if these data could be compiled and tabulated they would contribute some knowledge of what had been accomplished by different laryngologists with different operations. From this might be developed some sort of plan to follow in those cases of bilateral paralysis of the abductor muscles coming under the laryngologist's care. Third, I wanted to report these 2 cases for the consideration of the members of the academy.

To obtain my data, I sent out one thousand questionnaires to the members of the different national societies, and from these one thousand

questionnaires I received one hundred and twenty-one replies. Many of those who replied were most anxious to help by giving whatever information they had. I have compiled a set of charts, which may be of interest and which may substantiate some of the statements and conclusions made in this paper.

SUMMARY AND CONCLUSIONS

It is my belief that not all patients with bilateral paralysis of the abductor muscles need immediate operation. A patient, whatever the cause of his paralysis, with the cords in the cadaveric position, with a good voice and with no dyspnea on ordinary exertion may be watched until he seeks relief. Such a patient in confinement or under circumstances requiring excessive physical strain should be tracheotomized and operated on for paralysis of the abductor muscles at his convenience and that of the physician. From my study and experience, I believe it is wise to wait from six months to one year before operating unless it is definitely known that the nerves have been cut, because there have been reports of restoration of function after an elapse of eighteen months.

I feel that the surgical data gathered through my questionnaire prove without doubt that the greatest success in the treatment of bilateral paralysis of the abductor muscles is attained by those operations in which the arytenoid cartilage is attached either extralaryngeally or intralaryngeally. In view of the number of successful operations reported by Dr. King, it seems imperative that the King operation should be tried before any other extralaryngeal procedure. Arytenoidectomy, either through the King incision or through the wing of the thyroid cartilage, should be tried on one or both sides before resorting to an intralaryngeal operation. If the external operations on the arytenoid cartilages fail, intralaryngeal surgical procedure is in order, and I prefer the resection of the thyroarytaenoideus and the cricoarytaenoideus lateralis muscle after the method of Loré or Rawlins.[10]

If the laryngologist follows this sequence in the surgical treatment of bilateral paralysis of the abductor muscles, he will make no stabs in the dark, he will never do more than is necessary to give the patient a satisfactory result and he will most thoroughly conserve the interests of the patient.

140 East Fifty-Fourth Street.

10. Rawlins, A. G.: Operative Procedure for the Relief of Stenosis in Double Abductor Paralysis of the Larynx, Tr. Am. Laryng., Rhin. & Otol. Soc. **45**:414, 1939. Stevenson, W.: Submucous Cordectomy for Bilateral Abductor Paralysis: Report of a Case, Ann. Otol., Rhin. & Laryng. **46**:531-534 (June) 1937.

Case Reports

HEMORRHAGE INTO THE PARANASAL SINUSES FOLLOWING SUCTION DISPLACEMENT IRRIGATION

E. D. Furrer, M.D.; D. C. Stanard, M.D., and J. Kuykendall, M.D.
Eugene, Ore.

Proetz [1] in 1926 published his first work on the displacement method for the treatment of chronic inflammation of the accessory nasal sinuses. Since that time a voluminous bibliography has been accumulated on the results obtained by different authors, using various types of displacing fluids. Proetz [2] in 1937 reviewed the literature evaluating the displacement method.

Most authors agree that not all patients are amenable to the Proetz treatment and that careful consideration should be given in administering treatment by this method. We have been able to find only a few cases in which untoward results have occurred after this type of treatment. Gordon [3] cited a case in which strong massive suction was used and the patient suffered during the following two weeks from severe headache in the right frontal region. Operation revealed that the right frontal duct was plugged with herniation of polyps and mucous membrane.

Skillern,[4] in discussing one of Proetz's papers on the method of displacement, reported 2 cases in which attempts were made to empty the frontal sinus of a thick pustular discharge. The first case is the one cited in the preceding paragraph, which was reported by Gordon for Coates.[5] The other patient had had massive suction for several weeks. Radical opening of the sinus revealed punctiform hemorrhages throughout the thickened lining membrane. Both of these patients had been treated elsewhere.

The following report is one from our own experience in which a patient treated by the Proetz method revealed hemorrhage into the sphenoid and ethmoid sinuses at postmortem examination, although death was due to another cause.

1. Proetz, A. W.: Displacement Irrigation of Nasal Sinuses: A New Procedure in Diagnosis and Conservative Treatment, Arch. Otolaryng. **4**:1-13 (July) 1926.

2. Proetz, A. W.: An Evaluation of the Displacement Method with a Review of the Literature, Ann. Otol., Rhin. & Laryng. **46**:699-734 (Sept.) 1937.

3. Gordon, W.: Use of Negative Pressure in Otolaryngology: Report of a Serious Accident, Arch. Otolaryng. **16**:370-379 (Sept.) 1932.

4. Skillern, S. R., Jr.: Personal communication to the authors.

5. Coates, G. M.: Personal communication to the authors.

REPORT OF A CASE

A woman aged 23 years came to the office of one of us (D. C. S.) in January 1936, complaining of a chronic nasal discharge, from which she had suffered for several years. Examination revealed a postnasal drip and plugs of mucus high in the pharynx and posterior nares. Nasal ephedrine sprays were given at different times with moderate success. Displacement treatments were started on Feb. 14, 1938. Eight cubic centimeters of 0.5 per cent ephedrine in Locke's solution was used with a suction pressure of between 17 and 20 mm. of mercury.

The results obtained from this type of treatment were so satisfactory that displacements were given during the patient's subsequent visits to the office, which were on April 24, April 26, July 25 and July 27, 1938. She returned on June 5, 1939, complaining of pain in the right eye, which had been intermittent for a week. A tentative diagnosis was made of infection of the right frontal sinus, and a displacement treatment was given that day. The patient reported on her return, June 9, that numbness had developed in the right hand the day before but was subsiding at the time of her visit. Another displacement treatment was administered.

The patient was seen by one of us (J. K.) at her home about 5 p. m. the same day and at that time was unable to use the right arm or to know in which position it was being held. Examination revealed flaccid paralysis of the right arm and forearm; there was no loss of sensation or change in the tendon reflexes. Two hours later the paralysis included the right side of the face, the right hand and the right leg. She was conscious and could respond to touch. No other neurologic abnormalities were observed.

The patient was admitted to the hospital at 10:30 a. m. the following day. Examination revealed a temperature of 102 F., inability to talk, restlessness and anxiety. The fundi of the eyes appeared normal. The spinal puncture showed a pressure of 44 mm. of water. There were no cellular changes. The white blood count was 9,400, with 86 per cent segmented polymorphonuclear leukocytes, 5 per cent nonsegmented polmorphonuclear leukocytes, 1 per cent basophilic leukocytes, 7 per cent lymphocytes and 1 per cent monocytes. Muscular rigidity, opisthotonos and convulsions developed within a few hours after the patient's entrance into the hospital. That same afternoon cyanosis appeared and death occurred.

Postmortem examination revealed the following significant conditions: The left parietal-temporal lobe of the brain was soft, and a coronal section through this region showed an area 6 cm. in diameter undergoing encephalomalacia. The remaining portion of the brain revealed no distinctive pathologic changes. The venous sinuses about the pacchionian bodies on the left side contained what appeared to be adherent blood clots. The right sphenoid sinuses and the posterior ethmoid sinuses were completely filled with clotted blood.

Microscopic sections through the areas of infarction showed sharply demarcated borders of necrotic tissue. Microscopic sections through the dura and the walls of the superior longitudinal sinus revealed dense areas of fibrosis with scattered and collected lymphocytes. The blood clots about the pacchionian bodies were attached to the intima by proliferating fibroblasts and contained many polymorphonuclear leukocytes.

COMMENT

It seemed difficult for us at first to determine the exact sequence of events in this case. It appeared obvious, however, that the treatments given the patient before her return on June 5, 1939 had no bear-

ing on the subsequent history. At the time of her visit on June 5 she complained of having had pain in the right eye for a week. It is conceivable that the cerebral changes were beginning when the pain in the right eye occurred.

From June 5 until her death on June 10 the patient had had two displacement treatments. There was no bloody fluid aspirated from the sinuses during her treatment of June 9. This would indicate that no hemorrhage had occurred after the displacement of June 5.

Our first impression at the time of the necropsy was that an embolus, caused by either air or septic material, had been aspirated into one of the venules and that the infarction was due to embolism. We considered this point of view a little far fetched, although we believed that the hemorrhages into the paranasal sinuses were due to the suction from the last treatment.

The beginning organization of the thrombus in the venous sinuses of the dura, the fibrosis and the inflammatory reaction in the walls indicate that the pathologic changes must have anteceded the displacement treatment of June 9, 1939. These microscopic observations proved to us that the displacements, per se, had no direct bearing on the cerebral changes, since a much longer time than twenty-four hours is required to produce this type of histologic manifestation.

CONCLUSIONS

We have reported a case of hemorrhage into the paranasal sinuses following the use of the Proetz displacement treatment.

It is our opinion that the cerebral changes found at necropsy anteceded the last two treatments and were due to thrombophlebitis of the venous sinuses of the dura as the result of absorption of infected material from the venules about the paranasal sinuses which empty into these large draining venous channels.

The physician should recognize that, no matter how insignificant a type of medical or surgical treatment is, there is always the possibility that complications will develop, in which case an erroneous interpretation of the causes may be made unless careful study is given.

LARYNGEAL TUBERCULOSIS IN AN INFANT

SHIRLEY HAROLD BARON, M.D., NEW LONDON, CONN.

Tuberculosis of the larynx and trachea in very young children is considered rare. Such a disease process producing respiratory embarrassment in young children is almost unique. For these reasons, a case is being reported.

REPORT OF A CASE

A boy, C. F. M., 2 years and 8 months old, was admitted to the pediatric service of the Lawrence and Memorial Associated Hospitals on Aug. 17, 1938. He was suffering from moderate respiratory difficulty and had enlarged cervical glands.

Family History.—The child's mother had active pulmonary tuberculosis. Two months before his admission to the hospital, she had experienced a hemoptysis. Both of her parents had had tuberculosis.

Onset.—The child's illness began in February 1938 (seven months before admission), at which time he contracted bronchitis. After this he was more or less in ill health as a result of attacks of bronchitis. During the four months before admission, the cough had become aggravated; he also suffered from night sweats. Two months before admission, his mother noted swellings in the neck, which gradually increased in size.

Physical Examination.—He was a pale, fair-haired, light-skinned boy. He was not in acute distress but was breathing in a wheezy, moderately croupy manner.

Neck: Several large tender discrete palpable nodes were found anteriorly and posteriorly. The external jugular veins appeared full and slightly distended. This was exaggerated by coughing.

Lungs: There were bulging of the left anterior wall of the upper part of the chest and diminished expansion of the right anterior wall of the upper part of the chest. There was widened mediastinal dulness posteriorly in both directions, with bronchial breathing on auscultation over the spine. A prolonged expiratory phase could be elicited throughout both lungs, and dry musical rales were present throughout. These phenomena were more marked toward the apexes. There were no other significant physical manifestations.

Diagnosis.—On the patient's admission the pediatricians offered a diagnosis of tracheomediastinal and cervical lymphadenitis, probably tuberculous, with possible parenchymal involvement of the lung, tuberculous in nature. They expressed the belief that compression of the trachea or of the main bronchi accounted for the wheeze and the emphysematous breath sounds and that pressure on the veins of the neck caused the venous congestion.

On the first evening of the patient's stay in the hospital (August 17), I saw him and made the following notation: "There is no evidence of an acute inflammatory process in the ears, nose or throat. Discrete, palpable cervical nodes, larger on the left, are present. There is very slight retraction of the suprasternal notch on inspiration. Respiration is noisy—of the type which suggests a partial obstruction along the laryngotracheal passage. The accessory muscles of respiration

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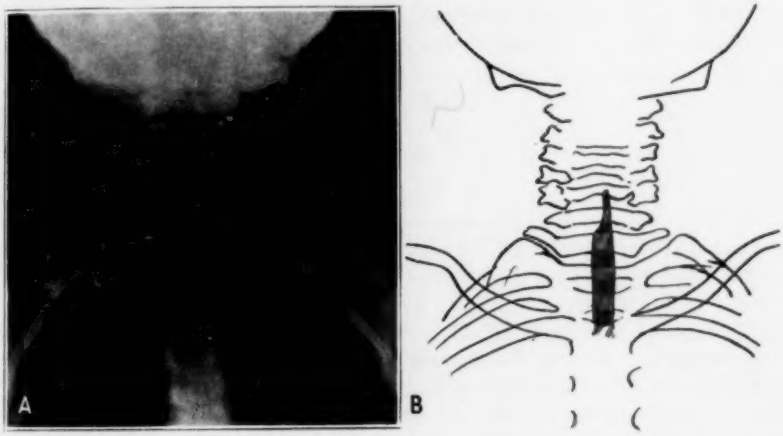
From the Ear, Nose and Throat and Pediatric Services of the Lawrence and Memorial Associated Hospitals.

are not brought into play. The patient is not cyanotic. The condition of the larynx can be determined only by direct laryngoscopic examination. The character of the voice, together with the other signs, suggests subglottic rather than glottic narrowing. Roentgen and laryngoscopic examination will determine whether there is endotracheal obstruction or extratracheal compression."

Roentgen Examination.—On the following day (August 18) a roentgenogram of the chest was taken. The plate showed clear pulmonary fields and enlarged peribronchial lymph nodes on the left side. A second roentgenogram (figure), taken at the same time in an attempt to visualize the tissues of the neck in detail, shows an interesting tracheal picture. The air space of the trachea is clearly visualized from the upper margin of the fourth thoracic vertebra to the middle of the sixth cervical vertebra. Its diameter from the fourth thoracic to the upper margin of the first thoracic vertebra is approximately 9 mm. At the latter point, the right tracheal wall begins to bulge, or fill in, so that the diameter of the tracheal lumen is but 4 mm. at the level of the middle of the seventh



A, roentgenogram showing sublaryngeal narrowing of the trachea, greatest near the larynx. *B,* diagrammatic drawing of the roentgenogram (*A*), accentuating the visibility of the tracheal narrowing.

cervical vertebra and 2 mm. at the sixth cervical vertebra. With the left tracheal wall nearly straight and the right indented, the air space appears as an inverted pen with the point toward the larynx.

Course.—For the first five days the course was fairly constant. The child's breathing was somewhat labored and noisy, with a croupy sound, and he was hoarse. Steam inhalations afforded no relief. In fact, the patient seemed better without them. On the sixth day the breathing became more difficult and laryngoscopic inspection was requested. On the seventh day I made a laryngoscopic examination of the patient and reported:

"There is a diffuse, red, turban-like swelling of the epiglottis. The mucous membranes of the arytenoids, the aryepiglottic folds and the ventricular bands are swollen and red. Near the posterior end (mesial surface) of the right vocal cord is a small elevation with superficial exudate. Granules (without exudate) are seen also on the left vocal cord. Diagnosis: Tuberculosis of the larynx."

The subglottic area could not be clearly visualized. In spite of the extensive inflammatory process of the larynx, the glottic space seemed ample for breathing.

The assumption was that the difficulty in breathing was due more to the subglottic swelling lower down, not seen on laryngoscopic inspection but noted in the roentgenogram.

The temperature, which ranged between 98 F. and 101 F. before laryngoscopic examination, rose to 103 F. after this procedure. It was 102 F. the second day and 101 F. the third day. On this day, the patient was dismissed from the hospital to a sanatorium.

Laboratory Examination.—A direct smear of material obtained from the larynx at the laryngoscopic examination revealed a single tubercle bacillus. Smears of material from the nose and throat were negative for Klebs-Löffler bacilli.

A guinea pig which was given an injection of urine from the patient died. Autopsy was not done, because the animal died too soon after receiving the injection for any reaction to have taken place. A guinea pig which was given an injection of feces from the patient also died. Examination of the urine on August 23 gave negative results.

Blood Counts: On August 17 the value for hemoglobin was 58 per cent. The red blood cells numbered 5,350,000 and the white blood cells 18,650 per cubic millimeter, with 61 per cent polymorphonuclears (55 per cent segmented and 6 per cent nonsegmented), 34 per cent lymphocytes, 1 per cent monocytes, 3 per cent lymphoblasts and 1 per cent eosinophils. The red cells showed marked hypochromia. On August 23 the white blood cells numbered 14,950, with 58 per cent polymorphonuclears, 42 per cent lymphocytes, 1 per cent monocytes and 1 per cent eosinophils.

Further Course.—On September 2 the patient was admitted to the Suffolk Sanatorium, at Holtsville, N. Y., where he died on October 13.

Communications from Dr. Edwin P. Kolb, superintendent of that institution, stated:

"In addition to an acute cervical adenitis with extreme enlargement of the glands and mediastinal adenitis, C. M. had an advanced pulmonary lesion. It is quite probable that he also had mesenteric involvement. Although, on account of his age it was impossible to secure a sputum specimen, tubercle bacilli were found in mucus taken from the stools. No autopsy was done."

"Physical signs in the chest at the time of admission were: noticeable dulness of the upper mediastinum, slight dulness, very intense breathing and rales throughout the right lung. About two weeks after his admission, physical signs indicated caseation and cavitation in the upper lobe of the right lung. His temperature varied from 101 to 105 F., the pulse rate averaged 140, and the respirations 28 to 30. Two days before his death, there was considerable blood in the stools. His abdomen was very much distended at the time of his admission and during his stay here."

"I feel sure that there is no question of the cavitation in the upper lobe of the right lung of the boy during his relatively brief stay here. I examined him both on admission and at the end of a couple of weeks and I feel that the physical signs were quite characteristic. . . . During the past few years we have had several small children, one as young as 20 months of age, with acute, adult, pneumonic type of tuberculosis with definite cavitation."

"The roentgenogram of the chest (at the Suffolk Sanatorium) was taken with the portable machine and because of the boy's marked dyspnea, is not a particularly good picture."

The roentgenogram of the chest taken at the Lawrence and Memorial Associated Hospitals on August 18 was made with the standard machine, and the

details were clear, showing no definite discernible tuberculous parenchymatous process at that time.

COMMENT

Even without the advantage of autopsy, the diagnosis of tuberculosis of the larynx seems certain. There is nothing, however, to support the diagnosis of tuberculosis of the trachea other than the interesting tracheal roentgenogram and the type of respirations. It is an assumption, of course, but not far fetched, that from the markedly involved tuberculous larynx direct extension to the tracheal mucosa occurred. It is a quite different process from that described by Myerson [1] in the report in which he demonstrated that tuberculous granulomatous swelling of the trachea occurs through direct extension from involved pulmonary parenchyma via a connecting bronchus.

One would be inclined to attribute the respiratory distress to compression by the tracheomediastinal lymph nodes as a result of the marked adenopathy. This was probably not the cause. The tracheal narrowing, as demonstrated in the roentgenogram, was sufficient to produce respiratory embarrassment.

Tuberculosis of the larynx is not usually considered a disease of childhood, much less one of infancy. Thomson,[2] who quoted Perrin, stated that, though not unknown in childhood, it is rarely encountered in persons under 10 years of age. Of 500 patients examined by Mackenzie,[3] only 1 was under 15 years of age. In 100 autopsies he found the larynx diseased once between the ages of 5 and 10, four times between 10 and 15, and sixteen times between 15 and 20. Recently Rubin and Galburt [4] thoroughly reviewed the literature on laryngeal tuberculosis in children. They found but 2 cases of children under 3 years of age: that reported by Heinze, of a child less than a year old, among 1,226 autopsies; and that of the youngest patient on record, an infant 3½ months old, reported by Nobécourt and Tixier in 1909. On postmortem examination of the latter patient, the larynx was found to contain granulation tissue and ulcerations. In a series of 100 cases of tuberculous children studied by Rubin and Galburt, the youngest child having laryngeal tuberculosis was 5 years of age. These writers expressed the belief that more tuberculous larynxes would be discovered if routine examinations of the larynx (by the indirect method) were made.

SUMMARY

The case of an infant 2 years and 8 months of age, who had tuberculosis of the larynx and was examined by direct laryngoscopic procedure, is presented.

The literature on the subject confirms the belief that tuberculosis of the larynx in very young children is extremely rare.

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1. Myerson, M. C.: Tuberculous Ulcerogranuloma of the Trachea and Bronchi, Ann. Otol., Rhin. & Laryng. **49**:177-198 (March) 1940.

2. Thomson, St. C., and Negus, V. E.: Diseases of the Nose and Throat, ed. 4, New York, D. Appleton-Century Company, Inc., 1937, p. 720.

3. Perrin, cited by Thomson and Negus.[2]

4. Rubin, H., and Galburt, S.: The Larynx of the Tuberculous Child, Arch. Otolaryng. **30**:421-428 (Sept.) 1939.

CALCIFICATION OF THE NASAL QUADRILATERAL CARTILAGE

HARRY C. ROSENBERGER, M.D., CLEVELAND

Previous to Feb. 9, 1940 my experience and the teaching of others had led me to believe that the septal quadrilateral cartilage was always cartilaginous and never osseous.

REPORT OF A CASE

On Feb. 9, 1940 I operated on L. F. G., a man aged 45, at Lakeside Hospital, for the relief of nasal obstruction.

The procedure contemplated was a submucous resection to correct a marked superior and anterior deviation of the nasal septum to the left. This deflection was posterior to and did not involve the septal tip, which was in its proper position in the columella. With the patient under local anesthesia, the usual curved incision was made on the left side, distal to the deflection, and the mucoperichondrium was easily elevated posteriorly as far as the maximum deflection. The incision was then deepened, with the intention of incising the quadrilateral cartilage and freeing the mucoperichondrium on the opposite side, preparatory to removing the cartilage. Contrary to all my previous experience, the knife failed to reach the opposite mucoperichondrium but instead came to rest on a smooth bony-hard surface in the depths of the cartilaginous incision. This bony surface offered firm resistance, which was encountered throughout the incision, which extended from the floor to a point close to the nasal roof, as the septal deflection was high.

As any one may imagine, this was unusual and disconcerting.

I then removed the sheet of incised cartilage, which was not especially thick and extended vertically from floor to roof and posteriorly only some 2 cm., where it thinned and ended. This cartilage lay against the bone like a slab of cheese in a sandwich.

The deflection still persisted, and the bony partition was still intact. Finally, however, by the use of a sharp mastoid curet the bony wall was penetrated near the roof, where it was thinnest, the mucoperiosteum on the right was elevated with difficulty, because of its adherence, and the bony deflection was removed with bone forceps.

The calcified quadrilateral cartilage was moderately thick, especially as it approached the vomer, and was not laminated except for the single layer of cartilage over its anterior, or distal, part on the left side. Posteriorly the calcified cartilage blended with the perpendicular plate of the ethmoid and was not distinguishable from it.

Thinking that perhaps this patient might exhibit additional evidence of early or advanced calcification of cartilaginous structures in other locations, I referred him for roentgen examination of the larynx. The radiologist, Dr. Farmer, reported that, although the laryngeal cartilages, notably the thyroid, showed definite evidence of calcification, yet in his opinion, these changes were within the average variation for the patient's age and sex. A lateral roentgenogram of the nose after operation

showed several small dense areas about the periphery of the septal tip, which undoubtedly represented depots of relatively heavy calcium deposit in the shell of quadrilateral cartilage remaining.

COMMENT

The point I desire to emphasize is that the septal deformity in this case was unlike the common one seen as the result of trauma, in which the septal tip is often displaced from the columella and in which, at operation, the component septal structures are found to be stratified and angulated, the whole impregnated with structures resulting from fibrous and osseous change.

Histologists state that the cartilages of the nasal septum, the cricoid cartilage and the lateral portions of the thyroid cartilage are hyaline in type. That the laryngeal cartilages undergo gradual calcification, especially in the male, is well known. Laryngologists who have had experience with the technic of laryngofissure are aware of the occasional ossification of the thyroid cartilage, at least in its midpart, which renders its bisection somewhat difficult. However, it must be mentioned in this connection that histologists consider the midpart of the thyroid cartilage to be elastic in type, whereas the thyroid alae, as mentioned before, are composed of hyaline cartilage. Whether this fact has a bearing on the calcification of the thyroid cartilage, I do not know. If both the quadrilateral and thyroid cartilages are hyaline in nature, then it is curious that calcification should affect the one and not the other.

Similarly, if these cartilages are of like nature it is equally strange that rhinologists, in performing septal operations, should not have encountered calcification and reported technical difficulties because of it, as have the laryngologists in the surgical treatment of the larynx. Conversely, nowhere in textbook or rhinologic literature have I found any mention of osseous change in the quadrilateral cartilage except in those common instances of trauma previously mentioned.

Clinical Notes; New Instruments and Technics

DIRECT ADENOIDECTOMY

LOUIS GUGGENHEIM, M.D., LOS ANGELES

The use of the La Force adenotome alone is inadequate for the complete removal of adenoid tissue. The curet, in most hands, strips mucosa and leads to the formation of scar tissue. In cases in which hearing is normal and ears uninvolved, it may not be essential that the meticulous removal of all nasopharyngeal lymphoid tissue be carried out, but in cases in which hearing is impaired, complete eradication of adenoid tissue is imperative.

Fig. 1.—Andy Love palate retractor.

This should be accompanied by dilatation of the eustachian tubes under direct vision.

At the Los Angeles Children's Hospital the following technic has given satisfactory results.

The instruments used are:

A. The Andy Love palate retractor.
B. The Gergoiye tubal dilators.
C. The Guggenheim lateral curtain punch.
D. The Guggenheim triangular forceps.
E. The Guggenheim curved scissors.

After removal of the adenoid with the La Force adenotome and the control of bleeding with tampons saturated with epinephrine hydrochloride, the soft palate is elevated with the Andy Love retractor until the entire nasopharynx is well visualized. Usually the eustachian tubes can be seen at once.

Into the right tube the Gergoiye right dilator is inserted, pressure being exerted for twenty seconds. Next, the left tube is treated in the same manner.

Behind each posterior faucial pillar will be found the lateral lymphoid curtain. This may be removed with the punch, or it may be grasped with the triangular forceps and cut away with the small curved scissors.

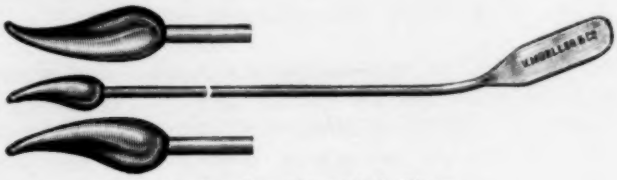


Fig. 2.—Gergoiye tubal dilator.

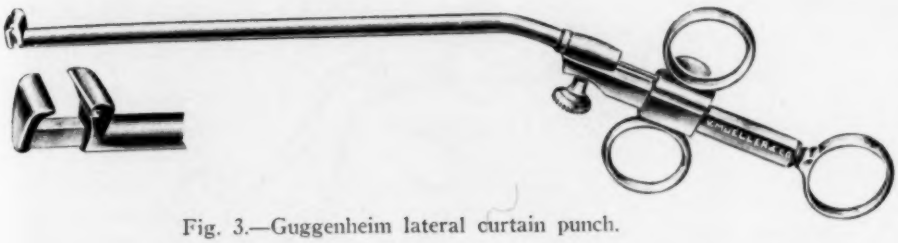


Fig. 3.—Guggenheim lateral curtain punch.

Fig. 4.—Guggenheim triangular forceps.

Fig. 5.—Guggenheim curved scissors.

After this, all remaining lymphoid tissue is removed with the triangular forceps, the scissors and the smallest La Force adenotome, under direct vision.

In the majority of patients below the age of 13 years who are without labyrinthine involvement, improvement in hearing may be expected.

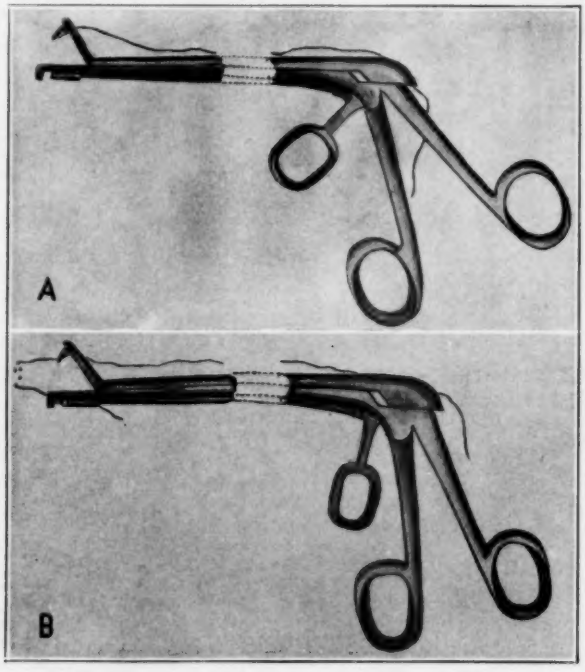
1033 Gayley Avenue.

This procedure was reported before the Research Study Club of Los Angeles in January 1939.

METHOD OF SUTURING THROUGH AN ENDOSCOPIC TUBE

E. I. Matis, M.D., Kaunas, Lithuania

The suture made through an endoscopic tube presents many difficulties, because the field of operation is limited and the spot which must be stitched lies at considerable depth. By using a new technic it is possible to find a solution of this problem. A new instrument for automatic suturing in the deep parts has been designed and constructed. This instrument is for use through a special endoscopic tube provided with a mechanism which enlarges its working lumen and gives more space for the manipulation of instruments. The new instrument (figure *A*) reminds one of an endoscopic forceps with a thin needle at the end and a finger piece at the handle.



Instrument for automatic suturing through an endoscopic tube: (*A*) in position for use and (*B*) after the suture has been pulled through.

The endoscopic tubes, especially favorable for manipulation with an endoscopic suture forceps, are of two types: (1) the expanding bronchoesophagoscope designed by me and (2) the endoscopic tube provided with a slot-shutter mechanism, which enlarges the operative field and allows easy manipulation of instruments.

The application of the suture is as follows: The expanding esophagoscope or the tube pivoted with the slot-shutter mechanism is introduced; the operative field

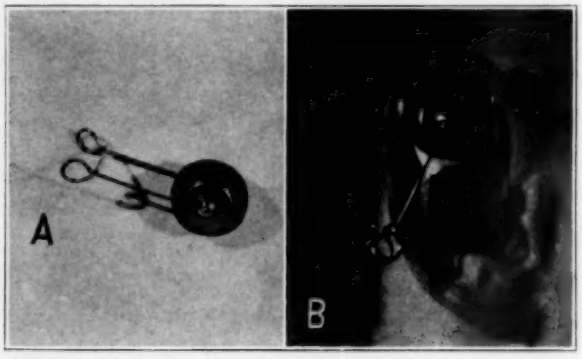
is maximally enlarged. The endoscopic suture forceps is introduced through the enlarged endoscopic tube and directed to the spot which has to be stitched. When the suture forceps is closed the needle passes through the spot which must be sutured. By pressure on the finger piece the silk or catgut is automatically drawn through; the instrument is then removed, leaving the suture ready to be tied (figure *B*).

Suturing at a depth through an endoscopic tube can be used for many purposes: for controlling hemorrhage, for the fixation of deep lying tissue, for the closing of a wound and generally for the endoscopic operation.

TREATMENT OF HEMATOMA OF THE EAR BY MEANS OF A NEW PRESSURE CLAMP

JAMES B. LOUNSBURY, M.D., ANN ARBOR, MICH.
Surgeon, University of Michigan Health Service and Athletic Association

Hematoma of the auricular pinna is an injury not commonly seen by the average practitioner. By those responsible for the care of athletes, more particularly of boxers and wrestlers, the injury is often encountered. Many physicians



Photographs showing: *A*, the Lounsbury clamp for cauliflower ears: *B*, the clamp in place.

are at a loss as to how to treat the patient. Treatment may vary from neglect to incision and drainage of the ear. Faulty care of the hematoma in the early stage may result in extreme disfigurement.

The treatment of the fresh hematoma is simple. Aspiration with a small needle and a Luer syringe under reasonably aseptic conditions and the application of pressure for forty-eight hours are all that is necessary. The procedure is to be repeated as often as needed until the formation of the serosanguineous material beneath the integument has ceased.

The method of applying pressure to the bleeding area is the enigma. Collodion, the use of which is often advised, has no rational qualification for use in the treatment of this condition. The massively padded cotton or gauze dressing with

an elastic bandage about the head is not only unsightly but often extremely uncomfortable. It is also difficult to know that pressure is being applied to the correct area.

After treating a large number of auricular hematomas by the pressure bandage method, I was led to design the illustrated clamp, which has given satisfactory results since its use was instituted. The clamp[1] consists of firm sponge rubber pads attached to a spring wire clip. After aspiration of the hematoma, a small sterile gauze pad is applied over the puncture wound and the clamp placed as shown in the illustration. It is left in place forty-eight hours. A few patients find the clamp uncomfortable when they are sleeping on a pillow, in which case they remove the clamp and reapply it the following morning. The need for repeated aspirations has been much reduced by the use of this simple device.

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1. The clamp may be obtained from the Penn Surgical Manufacturing Co., Inc., 1407 North Eighth Street, Philadelphia, under the name "Lounsbury clamp for cauliflower ears."

Abstracts from Current Literature

Ear

ETIOLOGY AND SURGICAL TREATMENT OF ACUTE MASTOIDITIS. H. MCCASKEY, J. LAWRENCE SIMS and R. E. ESTLICK, Bull. Am. Coll. Surg. **25**:179 (June) 1940.

The authors have made a survey of 320 cases of acute mastoiditis. The patients were treated at the Indiana University Medical Center from Jan. 1, 1934, to Jan. 1, 1939. The authors found that the average preoperative period of hospitalization was eight and one-half days, while the average total stay of the patient in the hospital was thirty-six days for children and eighteen days for adults. The average duration of the aural drainage before surgical intervention was twenty-three and one-half days. An analysis of the mastoid cultures taken either at the time of surgical intervention or at autopsy indicated that the streptococcic group of organisms caused 80 per cent of the attacks of the disease, the staphylococcic group 10 per cent and the pneumococcic group 6 per cent; the rest of the attacks were due to the usual less frequent organisms. In complications the streptococcus is the most frequent organism. The authors mentioned the usual symptoms and the importance of the use of roentgen and laboratory examinations, particularly of the Schilling blood count, in establishing a diagnosis.

They found a perisinal abscess in 27 cases (8.4 per cent), lateral sinal thrombosis in 19 cases (5.9 per cent), organismal meningitis in 17 cases (5.3 per cent), seromeningitis in 3 cases (0.9 per cent), septicemia in 21 cases (6.5 per cent), extradural abscess in 6 cases (1.8 per cent), petrositis in 5 cases (1.5 per cent) and Bezold's abscess and facial palsy each in 2 cases (0.6 per cent). The total mortality rate was 11.1 per cent (or 38 deaths), but there was only 4.9 per cent (17 deaths) in the operative cases.

Treatment was discussed both from the medical and from the surgical standpoint, not only for simple mastoiditis, but for each respective complication. While this discussion of treatment was in outline form, it was extremely conclusive and practical.

In conclusion the authors stressed the value of their statistics as an aid to early diagnosis and as an ultimate help in obtaining better therapy for mastoiditis.

PERSKY, Philadelphia.

OPERATION FOR THE CURE OF POSTAURICULAR FISTULAE: REPORT OF EIGHT CONSECUTIVE CASES. SAMUEL D. GREENFIELD, Laryngoscope **50**:312 (April) 1940.

Greenfield reports 8 consecutive closures of postauricular fistulas. He describes his procedure in detail and recommends the following steps for success: revision of the mastoid, lowering of the posterior wall of the bony canal, removal of the mastoid tip to permit the digastric muscle to present itself into the cavity, use of a temporal muscle flap to fill the mastoid cavity, and use of a skin flap slid forward to cover and close the cavity without tension on the postauricular sutures.

WOOD, Newark.

THE SIGNIFICANCE OF ACCESSORY SINUS DISEASE IN CHILDREN. ALFRED WACHSBERGER, M. Clin. North America **23**:681 (May) 1939.

Sinal infections in infancy and childhood hold a greater potential danger than such infections in adult life. Infection occurring during the period of pneumatiza-

tion of these structures may permanently arrest this process. Lack of normal pneumatization may lead to lifelong chronic infection.

Infection originating in the sinuses may attack structures (such as surrounding periosteum and bone) by direct local extension or by transmission by way of the lymph channels to the lower levels of the respiratory system. A common mode of extension is by continuity of tissue to the bronchi and lungs. Chronic bronchitis, frequently bronchiectasis and occasionally pulmonary abscess may be traced to nasal infections.

The mechanical importance of free nasal airways must be emphasized. Ventilation of the sinuses depends on alternate changes in air pressure, negative with inspiration and positive with expiration. Any interference with this mechanism may result in obstruction to the ostiums, retention of secretions within the sinuses and possible suppuration.

EUGENE H. SMITH, Ogden, Utah. [AM. J. DIS. CHILD.]

EXPERIMENTAL STUDY OF THE RELATIONS BETWEEN THE PUPIL AND THE LABYRINTH. G. ZANZUCCHI, Arch. ital. di otol. **52**:105 (March) 1940.

This study embraces the modifications of the pupil to sound stimuli, pupillary reactions to thermal stimuli and reactions to rotatory and galvanic stimulation. The experiments were conducted on pigeons, both before and after opening of the bony labyrinth, and the reactions were recorded in motion pictures. The results from sound stimulation (tuning forks and Edelmann's whistle) were the production of nystagmic eye movements, corresponding to the semicircular canal exposed, and mydriasis accompanied by hippus, which persisted after ablation of the cochlea but disappeared following cocainization of the labyrinth. It is thus evident that these pupillary reactions were due to acoustic stimulation of the otolithic and ampullar structures. The use of physostigmine salicylate, atropine and morphine in large doses caused disappearance of the reactions, which, on the contrary, were not modified by instillation into the conjunctival sac of a 1 per cent solution of cocaine with an equal amount of a 1 : 10,000 solution of epinephrine bitartrate.

Both hot and cold irrigations (with precautions to avoid cutaneous reflexes), applied through the auditory canal and directly to the uncovered semicircular canal, caused mydriasis, usually preceded by a temporary myosis. The dilatation was not prevented by cocainization of the tympanic cavity but did not occur when the labyrinth was cocainized or removed. Chilling of the labyrinth caused disappearance of the pupillary reflex to sound; warming tended to accentuate it.

On stimulation by rotation, the pupillary reactions consisted of contraction during the rotation, followed, when the turning was stopped, by a fugitive increase of the myosis and then by mydriasis and hippus. The phenomena were unchanged by opening of a semicircular canal or cocainization of one or both labyrinths. The reaction was even accentuated by complete removal of the membranous labyrinth, made twenty to forty days prior to the test.

With galvanic stimulation of the labyrinth the effect on the pupil was the production of mydriasis on closing and during the passage of the current, which was proportional to the intensity of the stimulus. The pupillary reactions persisted unchanged after anesthetization of the labyrinth. They became more intense immediately after ablation of the labyrinths and increased in intensity for some days; finally, as the degeneration of the vestibular nerve and centers proceeded, the reactions became progressively less until they disappeared altogether. It is noteworthy that after the labyrinths were cocainized or removed, the reactions to sound and thermal stimuli disappeared, while those to rotatory and galvanic stimuli remained and were intensified. This behavior of the pupil recalls the paradoxic phenomena of Langendorff from resection of the cervical portion of the sympathetic trunk and the superior cervical ganglion. The paradoxic excitability of the pupil to rotatory stimulation after removal of the labyrinth led Zanzucchi to study the effect of reflexes of the neck on the pupil. His experiments revealed that rotation of the body (the head being fixed) and twisting the neck

produced myosis, proportional to the amount of rotation; the reaction was not changed by removal of both labyrinths. The myosis, then, is a true pupillary neck reflex—a movement of the pupil associated with deviation of the eyes, itself a neck reflex. Since rotation produces a stretching of the muscles of the neck, the organism would react by contraction of the external muscles of the eye and of the sphincter of the pupil, the latter already rendered more excitable by the ablation of the labyrinth and the consequent degeneration of the nerve. This phenomenon may help to explain contradictory results encountered in vestibular tests in cases of disease of the ear and permit a more precise localization of the lesion.

DENNIS, San Diego, Calif.

LATENT OTITIS AND DISTURBANCES OF NUTRITION IN INFANTS. U. FERRI, Lattante **10**:143 (April) 1939.

There is a difference of opinion among pediatricians as to the importance of latent otitis media, mastoiditis and antral infection as a cause of severe gastroenteritis. In many cases operations on the antrum or on the mastoid have indicated that they were not the predominating factor in the nutritional disturbance. However, in a case of diarrhea which is running an acute or subacute course and is not responding to the usual dietetic and constitutional treatment, one should not hesitate to resort to operation as a possible means of relief.

HIGGINS, Boston. [AM. J. DIS. CHILD.]

OTITIS AND OTOMASTOIDITIS IN THE NURSLING. I. TOMA, MARIA GEORGESCU and V. PETRESCU-COMAN, Med. inf., Bucuresti **1**:277 (May-June) 1939.

Otitis is frequently encountered in nurslings in institutions. Thirty-seven and seven-tenths per cent of the nurslings hospitalized at the Institute for Puericulture have had otogenic infections. Otitis in the nursling can present different clinical aspects: acute otitis, subacute otitis, latent otitis and finally otomastoiditis. Forms of a great deal of importance from a diagnostic standpoint are subacute otitis, latent otitis and mastoiditis. Behind a picture characterized clinically by fever, grave gastrointestinal troubles, extensive loss of weight and the different signs of otitis, disguised or absent, there often occurs hidden or subacute otitis. In the course of latent otitis, signs of involvement of the ear may be absent; digestive troubles as well as temperature are variable in intensity and nature. The state of nutrition dominates the clinical picture. In otomastoiditis, the general condition of the patient, digestive disturbances (uncontrollable vomiting and diarrhea), high fever, catastrophic loss of weight and hyperleukocytosis are the chief elements in diagnosis. The clinical picture under which latent otitis presents itself may cause the condition to be recognized only at autopsy. In cases of such a clinical picture as has been described, it is preferable to practice repeated puncture of the drum than to allow otitis to develop with its serious consequences. In the course of otomastoiditis energetic medical treatment rarely gives satisfactory results. In no case ought one to temporize, awaiting results from this treatment. The pediatrician is more competent than the specialist to fix the time of intervention. This should be practiced with the region under local anesthesia and should be done rapidly.

FROM THE AUTHORS' SUMMARY. [AM. J. DIS. CHILD.]

THREE CASES OF CEREBELLAR ABSCESS IN ACUTE PURULENT OTITIS MEDIA. JOHN HYBASEK, Časop. lék. česk. **76**:549 (May 7) 1937.

Cerebellar and cerebral abscesses are much rarer with acute than with chronic otitis, as is shown by the statistics gathered by Kolmijcenko, in which in 85 per cent of cases the lesions followed chronic otitis. The cases cited are those of a man aged 42, a girl aged 7 years and a boy aged 16 years. In these the cerebellar abscess was diagnosed five weeks, three months and nine days, respectively, after onset of otitis. The extension in the first and third cases was through formation

of a perisinal abscess, periphlebitis and pachymeningitis. In the second case the infection traveled through the internal auditory canal. The patients in the first and third cases made apparently complete recoveries in seven weeks; the patient in the second case died of diffuse purulent meningitis.

STULIK, Chicago. [AM. J. DIS. CHILD.]

Pharynx

TREATMENT OF DISEASES OF THE THROAT. LYMAN G. RICHARDS, J. A. M. A. **115**:501 (Aug. 17) 1940.

The author briefly comments on the structures contained in the mesopharynx and the hypopharynx and sets forth the principles of treatment, dividing the latter into (1) systemic measures and (2) local measures.

With this plan, he discusses follicular tonsillitis, pharyngeal diphtheria, Vincent's angina, peritonsillar abscess, acute pharyngitis and retropharyngeal abscess, with especial emphasis on the technic of surgical procedures that may be indicated. The general plan throughout is sound, and every now and again some personal preferences are given.

In summarizing the chronic infections of the mesopharynx, the author considers the therapy of chronic tonsillitis. Tonsillectomy is the treatment of choice when the history and the conditions observed point to definite infection of the tonsils, but it is necessary to eliminate the question of tuberculosis and syphilis, the latter calling for systemic treatment and the former for complete removal. For simple chronic tonsillitis surgical intervention is better than diathermy. The presence of a malignant condition calls for irradiation with roentgen rays and radium. Foreign bodies in the mesopharynx should be removed either by indirect laryngoscopic procedure and use of grasping forceps or by direct endoscopic methods, which are far better.

Acute laryngitis requires the usual methods of therapy. The treatment of chronic laryngitis depends on the cause and requires the elimination thereof, if possible. The presence of tuberculous laryngitis calls for the general measures of constitutional care and for local treatment. The author prefers puncture cauterization of the tuberculous lesions. Tumors of the hypopharynx must be dealt with according to their position and the nature of the new growth. The treatment of these conditions is now being highly specialized and should be undertaken by those especially interested and extensively experienced in this type of work.

Foreign bodies in the esophagus should be removed by the esophagoscopic method; laryngeal paralyses must be cared for on the basis of the cause, in which regard conditions within the thorax should be corrected, if possible.

Disease of the central nervous system will entail many diagnostic difficulties. A careful and intelligent mirror examination of the larynx is the basic procedure on which almost all therapy will depend.

GORDON, Philadelphia.

INFLUENCE OF ADENOIDECTOMY ON THE CALCIUM-PHOSPHORUS-MAGNESIUM CONTENT OF THE BLOOD SERUM. C. CALABRESI, Arch. ital. di otol. **52**:273 (June) 1940.

Calabresi examined the blood serum of 28 children of both sexes, varying in age from 3 to 9 years and showing marked characteristics of adenoidism, in order to determine the influence of adenoidectomy on the content of calcium, phosphorus and magnesium. Examinations were made before operation and five days and twenty days afterward. The results of the examinations were: for calcium the average value before operation was 15.7 mg. per hundred cubic centimeters, five days afterward, 12.5 mg. and twenty days afterward, 11.5 mg.; for magnesium the figures were 3.5, 3 and 2.6 mg., respectively; inorganic phosphorus averaged 6.7, 5.2 and 5.4 mg. for the respective periods, and the averages for acid-soluble phosphorus were 8.5, 7.3 and 7.3 mg. The investigation thus reveals that the blood

serum of children with adenoids contains larger amounts of the three elements than are found in that of normal children, while after adenoidectomy there occurs a decrease in the amounts of all three elements, with a tendency to approach normal percentages.

DENNIS, San Diego, Calif.

STATISTICAL OBSERVATIONS REGARDING PAPILLOMA IN THE THROAT AND BUCCAL CAVITY. M. IKEMA and K. LI, Oto-rhino-laryng. **13**:115 (Feb.) 1940.

The authors report 9 cases of papilloma of the throat and buccal cavity. Six patients were males and 3 were females. Three of the patients were in the second decade of life, 4 in the third and 1 each in the fourth and fifth decades. Only 1 of the 9 used tobacco and indulged in alcoholic drinks. There were 4 with chronic rhinopharyngeal infection and 2 with acute infection.

The papilloma originated from the uvula in 5 cases, from the hard palate in 2 and from the tonsil in 2. These tumors had the appearance of a mulberry, were round or oblong and white or light pink, and varied from the size of a flaxseed to that of a chestnut. None of the 9 patients was aware of the presence of the tumor.

The tumors were removed from their stumps, under local anesthesia, with the scissors.

The authors collected from Japanese literature 52 cases of papilloma of the pharynx. The patients included 30 women and 22 men. There were 15 papillomas of the buccal cavity, 9 of which were in men and 2 in women; in the case of 4 there was no mention of the sex. There were 22 growths of lingual origin, 13 being in men and 9 in women.

HARA, Los Angeles.

Nose

THE FUNCTIONAL POINT OF VIEW IN RHINOLOGY. ARTHUR W. PROETZ, J. A. M. A. **115**:421 (Aug. 10) 1940.

Proetz, in his chairman's address before the Section on Laryngology, Otology and Rhinology at the Ninety-First Annual Session of the American Medical Association, New York, June 13, 1940, discusses in moderate detail the fundamental principles of certain rhinologic concepts which are sound and rational.

Many specialists, as well as physicians in the fields of internal medicine and general surgery, have turned to the realization that, in the ultimate, it is the function of any given structure that counts.

In the field of rhinology, the distinguished author once more emphasizes, as he has done before, the importance of knowing how a nose works. He pleads again and again for the need of due reflection on this, with a view to conserving the function of the organ. He deplores the small amount of space given in standard textbooks and monographs to nasal physiology, citing the scarcity of material available when one attempts to prepare a course of lectures to advanced students. He points out the pitfalls that beset the rhinologic surgeon unless he ponders well the functional viewpoint.

He makes brief comments on the differentiation between a sinal empyema and an abscess. He recalls the attempt in recent years to deal with intranasal conditions structurally instead of functionally, so that often nasal tissues were sacrificed and air currents were thus misdirected against certain sensitive portions of the upper part of the nose, with the production of intractable headaches. Much crusting resulted from too extensive surgical interference, in many instances, and this led the surgeon to believe that he had eradicated incompletely, with the result that "more and more was done—instead of less and less—for the point of view was structural instead of functional."

The author modestly admits his plea many years ago against the indifference that some of the foremost rhinologists showed toward a finer appreciation of nasal physiology, but he is happy that many rhinologists have investigated these fields throughout recent years, with the grand result that American laryngology has made splendid progress, though due credit is given to the fine work accomplished in England, in Germany and especially in the north countries.

He speaks of new work that was to have been discussed at the fourth International Congress of Otolaryngology, to have been held in Amsterdam but "now regrettably postponed," this new work coming almost exclusively from the laboratories of rhinologists actively engaged in clinical work. The application of such knowledge, he states, is twofold: "To heed what has been done in the laboratory and to apply it wisely to the patient: to adopt new methods, new drugs, new operations intelligently is progress."

Old methods, old drugs and old operations, when they have been discredited, should be abandoned.

The author concludes this address with the following: ". . . I wish only to point out that the functions of the nose, whatever they may ultimately prove to be, have a vital influence on the air we breathe. What could be more important?

"I wish to add that it is not enough to maintain breathing channels but that the mechanisms by which the nose keeps itself clean must be conserved or restored if it is to continue functioning and maintain its possessor in comfort. It is not enough to know that quantities of moisture are required to saturate inspired air unless we act on that knowledge by conserving and cultivating the humidifying equipment of the mucosa, already often taxed to the limit by the droughts of central heating. It is not enough to know that air currents are evenly distributed over the streamlined contours of the nasal fossae without being keenly aware that they may not be misdirected with impunity. . . . Little by little, as the physiology of the nose is better understood, therapeutic and surgical methods will conform to it. . . ."

GORDON, Philadelphia.

Miscellaneous

CONGENITAL CRANIO-FACIAL MALFORMATION (PTEROSCHISIS, ENCEPHALOCELE AND HYPERTELORISM). O. ARMENIO, Arch. ital. di otol. **52**:251 (May) 1940.

Armenio reports a case of congenital deformity observed in a boy aged 3 years and represented by dehiscence of the right lateral wall of the nose, flattening and broadening of the root of the nose and bilateral frontal encephalocele. The two inner canthi were 55 mm. apart. The Wassermann reaction was negative. The nasal bones could be palpated, and the interior of the nose was normal. The malformations are said to be due to a fault of development of the precordial cartilage, dependent on a hypophysial, endocrine dysfunction initiated during intrauterine life.

DENNIS, San Diego, Calif.

CLINICAL SIGNIFICANCE AND VALUE OF STUDY OF THE BASE OF THE SKULL. W. LOEPP, Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:291 (March) 1940.

The author believes that axial roentgen examination of the base of the skull is of importance from both a diagnostic and a therapeutic standpoint.

His technic permits a visualization of the entire base of the skull from the ethmoid sinuses to the foramen magnum and laterally from the maxillary angle of one side to that of the other. In consequence, changes, such as sclerosis, atrophy, ulcerations of the bone and swelling of the soft tissues are easily identified.

He reports a series of cases in which there were lesions in the sinus or the epipharynx which were identified with extensions to the base of the skull. Included among the lesions were a number of carcinomas of the epipharynx, a fibroma of the superior maxillary sinus with erosion into the skull and a metastasis to the skull from an unsuspected and undiagnosed hypernephroma. The character of the findings acted as a guide to the therapeutic procedure, since surgical intervention alone was indicated in certain cases, while others required supplemental high voltage roentgen therapy.

In his conclusions he stresses the importance of this method of examination and emphasizes its early use for any lesion about the epipharynx, to facilitate both an earlier diagnosis and an earlier institution of more suitable treatment.

PERSKY, Philadelphia.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

ROBERT H. GOOD, M.D., *President*

WALTER H. THEOBALD, M.D., *Secretary*

Regular Monthly Meeting, Nov. 4, 1940

Labyrinthitis and Meningitis with Recovery: A Case Report. DR. WILLIAM E. GROVE and DR. GERHARD D. STRAUS.

A girl 7 years of age suffering from labyrinthogenic meningitis was cured by radical mastoidectomy and massive doses of sulfanilamide without surgical treatment of the labyrinth. In the first twenty-four hours following operation 140 grains (8.4 Gm.) of sulfanilamide was given; this was decreased to 60 grains (3.9 Gm.) daily for the next four days, then to 40 grains (2.6 Gm.) daily for eighteen days. After the first twenty-four hours the concentration of sulfanilamide in the blood was 23.8 mg. per hundred cubic centimeters and was maintained thereafter at from 12 to 16 mg. Two transfusions of citrated blood were given postoperatively. By the eighth day the spinal fluid cell count had decreased from 3,900 to 31, and it then gradually returned to the normal.

Physical examination eight months after operation showed the postauricular wound healed. A small amount of mucoid discharge was present in the middle ear. Functional tests showed that whispered voice was heard by the left ear at a distance of 2 inches (5 cm.) and by the right ear at 20 feet (610 cm.) and that spoken voice was heard by the left ear at 12 feet (366 cm.) and by the right ear at 60 feet (1,829 cm.).

Attention is directed to two unusual features in this case. First, it is a case of labyrinthogenic meningitis in which recovery occurred without labyrinthectomy. Second, despite apparent bacterial invasion of the leptomeninges via the inner ear, the patient has a functioning labyrinth. It is suggested that it may be advisable to procrastinate with respect to surgical treatment of the labyrinth until chemotherapy has been given a trial.

DISCUSSION

DR. W. E. GROVE: I once heard an otologist ask the question "How can you tell when a serous labyrinthitis becomes purulent?" The answer was "I do not know." I have to confess that this is also my position. We do not know whether this was a serous labyrinthitis which had become purulent. Certainly the labyrinth was not dead, although we could not get any response to cochlear stimulation. The child was too sick to tolerate many labyrinthine tests, and we do not know whether vestibular function was out at that time. We made as thorough a search as possible for a pathway into the meninges and were unable to find any lead. Because there were definite labyrinthine signs, we felt that the infection traveled through the labyrinth into the meninges and produced meningitis.

We are frank to say that we do not understand a number of things about this case, but we think it interesting that under the chemotherapeutic attack the cochlear function, which to the best of our knowledge was gone completely, returned to a considerable degree, as shown on the audiogram. This brings up the question whether past ideas about surgical intervention in labyrinthine disease must be changed somewhat in the light of the modern use of therapeutic drugs.

DR. ALFRED LEWY: Another has been added to the growing list of cases in which meningitis has been successfully managed by modern chemotherapy. I should

say that this case cannot be classified as one of labyrinthogenous meningitis. In the Weber test the sound was referred to the ear which had been operated on. This indicates that there was some function in that labyrinth, and the subsequent return of a certain degree of hearing further indicates that the labyrinthitis was in all probability serous and not suppurative. Failure to find a direct avenue of connection with the meninges occurs frequently in surgical operations on the mastoid process even when there is no question as to involvement of the labyrinth, so the absence of such an avenue gives no real information.

I think the great question is brought out by Dr. Grove as to how far chemotherapy is going to supplant surgical treatment of otogenic meningitis. Before the days of sulfanilamide patients with hemolytic streptococcus infection were never expected to recover, and few did either with or without surgical treatment. In other words, surgical intervention did not do any particular good in otogenic meningitis. Now it is a usual occurrence for patients with this disease to recover under chemotherapy. I believe the question as to whether the labyrinth should be operated on should be extended to that of whether the mastoid should be operated on. I know of several patients in whom otogenic meningitis was produced by unquestioned otitis media, and no other cause was found; yet these patients recovered without surgical intervention. I am not decrying the use of surgical procedure if there is any definite evidence of breakdown of the mastoid process or if the infection has gone on for two or three weeks, even though a breaking down of the bone cannot be diagnosed. It is probable that extension is taking place through the mastoid process, and I believe that in a case such as this surgical intervention is indicated. I know of a case in which the period between the appearance of the first symptoms of otitis media and the onset of meningitis was nine days, and the patient recovered without surgical treatment.

Otologists are, therefore, not yet prepared to answer the question of the relative values of the two types of treatment. They have not had enough cases in which they determined not to operate. But I believe the question will require an answer sooner or later.

Dr. Francis Lederer: Many things that are interesting at the time to the person who observes a patient are lost when the case is reported later. I believe it was said that "most surgeons report the cure of labyrinthitis by labyrinthectomy." I believe that statement covers too much ground. I do not think labyrinthine operations are done on all patients who show nystagmus. At least that is not my policy. There is one factor to be borne in mind in this case. The patient was a young child, and prognostically children offer a better outlook in meningitis than do adults. It is also possible that you were dealing with a circumscribed type of meningitis near the cisterna, and infections of this type often remain circumscribed for a long time. I understood that in the Weber test the sound was lateralized to the affected ear and that other functional tests were not done. Therefore much about the case remains rather vague.

The history of the patient and the finding of granulations as described might bring up two thoughts—i.e., that this was a Streptococcus mucosus type of infection or that it was an acute exacerbation of chronic suppuration. The thought also occurred to me—was not this really a middle ear process, and were indications present for a radical mastoidectomy? It is possible that an atticoantrotomy would have sufficed, and in view of the priority for the successful outcome which has been given to sulfanilamide, is it not possible that the operation deserves credit for the result? The sulfanilamide was given postoperatively, and it is difficult to say that a patient with 3,900 cells in the spinal fluid would not have recovered with an atticoantrotomy or a radical mastoidectomy. I believe that otologists particularly in reporting a case in which operation has been done have to guard against giving direct credit to drugs for the results obtained instead of admitting that a focus has been eradicated and proper drainage established and that therefore the operation had something to do with the recovery.

DR. THOMAS C. GALLOWAY: I like this definition of serous labyrinthitis: "If the function of the labyrinth is restored, the patient had a serous labyrinthitis; if not, it was suppurative." I understood from the report that only one hearing test was made. This test may be an important point of differentiation and should be repeated frequently.

Two weeks ago I had a patient with a pneumococcic infection of the ear, marked dizziness, nystagmus to the opposite side and a nonreactive labyrinth. For four days there was no hearing with the opposite ear masked, but on the fifth day the spoken voice could be heard at 1 foot (30.5 cm.). A complete simple mastoidectomy was done, sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) was given and the patient has improved rapidly. If one makes hearing tests often, usually in serous labyrinthitis at some time the cochlear function will be found present.

One question that may be important is, How far, if at all, can invasion of the membranous labyrinth go with resolution? About two years ago a French author attempted to show by histologic studies that recovery could occur in a slow process even when there was invasion.

DR. W. E. GROVE: Dr. Lewy's statements with reference to the question of operating or not operating on patients with otogenic meningitis interests me very much, but in spite of the decided benefit of chemotherapy I do not believe I should have the courage to withhold operation in a case of the type described. In spite of the fact that the best methods of examination were at our disposal, I do not feel that I know whether there was bone disease in this case. It was a clinic case, and as usual there was considerable difficulty in getting an accurate history. As Dr. Lewy said, granulations were within the tympanum, possibly indicating chronicity, but I do not think I should have the courage not to operate as widely as possible, and therefore a radical operation was done.

Dr. Lederer questioned whether the cure came from the operation or from chemotherapy. I do not know. I think possibly it came from both, but as Dr. Lewy said, prior to the use of chemotherapy cure in these cases was extremely rare, and for that reason I attribute the cure more to chemotherapy than to operative intervention. I do not believe this meningitis was localized. If it were, I should not expect to get a culture of hemolytic streptococcus in the spinal fluid removed by lumbar puncture. I think with Dr. Galloway that this probably was serous labyrinthitis. I did not state that it was purulent labyrinthitis, although I did state that in our opinion the infection went through the labyrinth to reach the meninges, for we were not able to find any other source of infection. I admit that our methods of treating infections through operative procedures are not so effective but that we might have missed a lead. How extensive an infection of the labyrinth can be followed by recovery I do not know. One cannot always be sure that there has been a purulent invasion of the labyrinth. When one says that a serous labyrinthitis is present, one cannot always be sure that it is not a purulent infection that has been taken care of. It is assumed that if a labyrinth recovers function there has been serous labyrinthitis and not bacterial invasion. I do not know that that question has ever been absolutely solved.

Chemotherapy in the Treatment of Complications of Acute Suppuration of the Middle Ear. DR. JOHN R. LINDSAY.

The material presented in the paper is limited to two types of complication, suppurative meningitis and suppuration in the petrous pyramid. Ten cases were presented, 8 of patients treated during the fall and winter season of 1939-1940, the other 2 of patients treated a year previously. There were 7 cases of diffuse meningitis and 1 of localized cisternal meningitis. In 4 the meningitis was secondary to suppuration of the petrous pyramid and in 4, to mastoid suppuration. Two additional cases of suppuration of the petrous pyramid were included. Charts were presented, showing the clinical progress of each patient and the details of

therapeutic measures. In the 10 cases there were 2 fatalities, 1 from pneumococcus type III meningitis secondary to petrositis and 1 from cisternal meningitis secondary to mastoid suppuration and sinus thrombosis.

The material afforded definite evidence on the relative places of surgical procedures and chemotherapy in the treatment of these severe complications.

1. In many cases of otogenic meningitis secondary to suppuration in the mastoid process it is possible to obtain a cure by chemotherapy without surgical intervention.

2. In cases in which there is no evident involvement of the petrous pyramid and in which the mastoid appears relatively small, the chances for cure with chemotherapy alone seem greatest. However, the inability to rule out such conditions as localized foci of necrosis and extradural or subdural abscess seems to indicate that the safest course at present is to use chemotherapy and surgical procedures combined. This applies only to cases in which there is clinical evidence of mastoid suppuration.

3. In cases of suppuration of the mastoid process and of the petrous pyramid in which there is a large focus of necrosis in the bone or an extradural or subdural abscess, a cure usually cannot be achieved with drugs alone in concentrations so far used.

4. The efficacy of chemotherapy appears to depend to a great extent on anatomic conditions. Extensive pneumatization provides anatomic conditions least favorable to chemotherapy, namely, collections of purulent exudate in large air cells and breakdown of cell partitions to form large necrotic foci.

5. Extensive pneumatization is associated with extension of the air cell system in and around the pyramid. Therefore, the anatomic condition least favorable to chemotherapy also predisposes to suppuration of the petrous pyramid.

6. Moderate concentrations of drugs probably lower the incidence of surgical treatment but have little influence on patients with a combination of virulent infection and extensive pneumatization.

7. Signs of toxic effect of the drug were produced in several instances, but in no case were they sufficient to interfere with treatment.

DISCUSSION

DR. GEORGE LIVINGSTON: After such a thorough dissertation there is little to discuss regarding the individual features of any case. I should like to supplement the account of Dr. Lindsay's experience with additional cases from Children's Memorial Hospital, which may have some bearing in so far as statistical material is of value. The value is limited, because the number of cases is still not large enough to enable one to draw final conclusions concerning the effectiveness of chemotherapy in otitis media at an early stage. There is no question of the value of this treatment in serious intracranial complications.

With a view to estimating the value of chemotherapy before complications have occurred, my associates and I collected in the outpatient department from January to June 1940 a series of cases in which the disease was in an early stage. This differs from a series of hospital cases, in which patients may be admitted with complications. The series was divided into approximately equal parts. Of 332 patients, 178 had chemotherapy: those with hemolytic streptococcus infection received sulfanilamide; those with pneumococcic infection, sulfapyridine; 154 were treated without chemotherapy. The figures are not impressive, and the differences between the two groups are not great; nevertheless, they may be of significance if the treatment is carried out in various other clinics and the results uphold these findings. The duration of otitis media in the patients from whom chemotherapy was withheld was 19.5 days and in those who were treated, 14.5 days. Mastoid complications occurred in 7.8 per cent (12) of those treated symptomatically and in 2.8 per cent (5) of those who received chemotherapy.

These figures tend to favor the action of sulfanilamide in early stages of otitis media. On the other hand, among hospital patients, many of whom came with frank mastoiditis, the majority did not have sulfanilamide, but some did have and are included in the outpatient group. In 1939, 91 operations on the mastoid were done. Nineteen of the patients operated on had received adequate chemotherapy. This does not prove anything except that mastoiditis can and in some cases does develop in spite of chemotherapy. In ten months of 1940, of 112 patients on whom mastoid operations were performed, 35 had adequate chemotherapy—approximately the same percentage.

If one is to engage to give sulfanilamide or sulfapyridine in the early stages of disease, adequate doses should be given immediately in severe cases. My associates and I have found some patients who responded favorably and others who did not. If there is no response in three days, we stop the administration of the drug. If there is a response, we have an additional problem, because we know that to stop medication before the disease is completely cured may lead to a recrudescence and leave the patient in a more precarious condition. I should like to emphasize one feature in the use of this drug. The leukocyte count usually drops. In our experience there are two significant drops. Usually the leukocyte count falls rapidly to the normal and remains there. We have found a few cases in which the count reached the normal level and then continued to drop. Once committed to chemotherapy, one has to continue it until the temperature is normal, the ear is dry and the patient is entirely well. The leukocyte count may continue to fall even as low as 1,500, with marked diminution in granulocytes. Sometimes in ten or twelve days, when signs of mastoiditis appear, the patient has a low leukocyte count and is less able to combat infection. That is not an argument against chemotherapy, but it is a factor that must be considered. My associates and I are inclined, if there are signs of complications, to operate somewhat earlier on patients treated with chemotherapy than on the others. We feel less confident in letting the infection go on, because we cannot evaluate the clinical picture so well.

Dr. Meyer S. Fox: Dr. Lindsay and Dr. Livingston have covered the subject of chemotherapy in meningitis and otitis media. I wish to present an additional case of acute otitic meningitis, which presented some interesting clinical problems.

The chart illustrating the clinical course is self explanatory. A child 6 years of age, weighing 60 pounds (27 Kg.), was admitted to the hospital with bilateral otitis media, the ear drums having ruptured spontaneously and drained for one week. He was placed on a regimen of azosulfamide[1] 45 grains (2.9 Gm.) daily, and this dose was continued for six days, at the end of which time the clinical course showed decided improvement and the right ear had become dry. The administration of the drug was discontinued, and after three days the patient was about to be discharged when headache, vomiting and subsequent signs of meningitis developed. The temperature was 104.4 F. The spinal fluid contained 2,000 cells per cubic millimeter, 95 per cent of which were polymorphonuclears, and direct smear and culture of the fluid were positive for hemolytic streptococci. A simple mastoidectomy was performed on the left side, and chemotherapy was started. The patient was given 60 grains (3.9 Gm.) of sulfanilamide orally daily for four days; with improvement in clinical symptoms this was reduced to 45 grains (2.9 Gm.) daily. The blood concentration was 5.6 mg. per hundred cubic centimeters. The spinal fluid became more nearly normal, and the clinical course continued to improve. After one week the sulfanilamide was reduced to 30 grains (1.9 Gm.) daily. Fifteen days after the onset of the meningitis there was an abrupt recurrence of the meningitic picture and the spinal fluid findings, the cell count rising to 1,775. The mastoid wound was explored, but no significant conditions were observed.

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1. Azosulfamide is disodium 4-sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxynaphthalene-3,′6′-disulfonic acid. This substance has been known as prontosil soluble, as prontosil and as neoprontosil.

It was felt that the activation was due to the drop in the dosage of sulfanilamide. My associates and I therefore increased the child's intake of drugs to 60 grains (3.9 Gm.) of sulfanilamide and 60 grains (3.9 Gm.) of azosulfamide daily, which resulted in a blood level of 8.5 mg. per hundred cubic centimeters. This dosage was continued for twelve days, and it was then reduced gradually during the next four weeks, during which the clinical course gradually improved and the spinal fluid became more nearly normal. There were several periods when the temperature was elevated. Supportive measures were used, and during the patient's stay in the hospital it was necessary to give eleven transfusions, varying between 250 and 300 cc., in order to keep the hemoglobin content of the blood up to 12 Gm. per hundred cubic centimeters.

This case shows the tendency for remission and exacerbation of meningitic symptoms to occur when the administration of sulfanilamide is stopped or the dosage is cut rather abruptly, the necessity for giving adequate amounts of the drug in order to control the clinical course and the depressing effect of the drug on the hemoglobin content of the blood.

Book Reviews

Directory of Medical Specialists Certified by American Boards, 1939. Directing editor, Paul Titus, M.D. Price, $5. Pp. 1,588. New York: Columbia University Press, 1940.

This volume is an official directory of the various American boards and in it are listed the names, addresses and biographic data of more than 14,000 specialists certified to date by these boards. The directory was authorized by the Advisory Board for Medical Specialties at its meeting in Chicago, in February 1939. The advisory editors for the directory are representative members of each of the American boards so far established and now functioning as such.

The directory is divided into sections. The Advisory Board for Medical Specialties, its subdivisions and personnel are first considered. Its organization and purposes are then explained.

In subsequent sections, the American Board of Anesthesiology, Inc., the American Board of Dermatology and Syphilology, the American Board of Internal Medicine, the American Board of Obstetrics and Gynecology, Inc., the American Board of Ophthalmology, the American Board of Orthopaedic Surgery, Inc., the American Board of Otolaryngology, the American Board of Pathology, the American Board of Pediatrics, Inc., the American Board of Psychiatry and Neurology, Inc., the American Board of Radiology, Inc., the American Board of Surgery and the American Board of Urology, Inc., are described as to historical data, personnel, procedures, requirements and functions.

At the end of each section are the biographic data of the diplomates who have qualified in the board under consideration.

The last three hundred pages of the book are devoted to an alphabetical list of all the diplomates who have met the requirements of any of the American boards. This list gives the name and address of each of the physicians and the boards in which he has qualified.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Place: Cleveland. Time: June 2-6, 1941.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Frank Brawley, 30 N. Michigan Ave., Chicago.
President-Elect: Dr. Frank R. Spencer, Physicians' Bldg., Boulder, Colo.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 19-24, 1941.

American Broncho-Esophagological Association

President: Dr. Gabriel Tucker, 250 S. 18th St., Philadelphia.
Secretary: Dr. Paul Holinger, 1150 N. State St., Chicago.
Place: Cleveland. Time: June 3, 1941.

American Laryngological Association

President: Dr. Gordon Berry, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Charles J. Imperatori, 108 E. 38th St., New York.
Place: Atlantic City, N. J. Time: May 28-30, 1941.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles.
President-Elect: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Ambassador Hotel, Los Angeles. Time: June 16-18, 1941.

SECTIONS:

Eastern.—Chairman: Dr. N. S. Weinberger, Robert Packer Hosp., Sayre, Pa.

Southern.—Chairman: Dr. William G. Kennon, Doctors Bldg., Nashville, Tenn.

Middle.—Chairman: Dr. Walter H. Theobald, 307 N. Michigan Ave., Chicago.

Western.—Chairman: Dr. Robert C. Martin, 384 Post St., San Francisco.

American Otological Society

President: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Secretary: Dr. Isidore Friesner, 36 E. 73d St., New York.
Place: Atlantic City, N. J. Time: May 26-27, 1941.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.